24jul03 09:48:37 User208600 Session D1585.1

SYSTEM:OS - DIALOG OneSearch
File 155:MEDLINE(R) 1966-2003/Jul W3 (c) format only 2003 The Dialog Corp.
File 5:Biosis Previews(R) 1969-2003/Jul W3 (c) 2003 BIOSIS

Set Items Description
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Ref Items Index-term
E1 1 NEUROTOXCITY
E2 4 NEUROTOXEMIA
E3 0 *NEUROTOXI
E4 1 NEUROTOXIA
E5 17181 NEUROTOXIC
E6 1 NEUROTOXIC A
E7 1 NEUROTOXIC A-BETA OLIGOMER
E8 1 NEUROTOXIC A-BETA PEPTIDE LOCALIZATION
E9 16 NEUROTOXIC ACTION
E10 1 NEUROTOXIC ACTION MECHANISMS
E11 4 NEUROTOXIC ACTIONS
E12 3 NEUROTOXIC ACTIVITIES
E13 12 NEUROTOXIC ACTIVITY
E14 1 NEUROTOXIC ACTIVITY NEUTRALIZATION
E15 1 NEUROTOXIC ADMINISTRATION
E16 1 NEUROTOXIC ADVERSE EFFECTS
E17 10 NEUROTOXIC AGENT
E18 1 NEUROTOXIC AGENT EXPOSURE
E19 1 NEUROTOXIC AGENT INTOXICATION
E20 8 NEUROTOXIC AGENTS
E21 1 NEUROTOXIC ALDEHYDE
E22 1 NEUROTOXIC ALKALOID
E23 1 NEUROTOXIC ALKALOIDS
E24 1 NEUROTOXIC ALPHA-LATROTOXIN
E25 1 NEUROTOXIC ALZHEIMER AMYLOID FRAGMENT BETA25-3
E26 1 NEUROTOXIC AMGYDALA LESION
E27 1 NEUROTOXIC AMINO ACID
E28 1 NEUROTOXIC AMYGDALA LESIONS
E29 1 NEUROTOXIC AMYLOID
E30 1 NEUROTOXIC AMYLOID BETA PEPTIDES
E31 3 NEUROTOXIC AMYLOID FIBRILS
E32 1 NEUROTOXIC ANATOXIN-A
E33 1 NEUROTOXIC ANTIRETROVIRAL THERAPY
E34 3 NEUROTOXIC ASPECTS
E35 1 NEUROTOXIC BASOLATERAL AMYGDALA LESIONS
E36 1 NEUROTOXIC BETA-A4 AMYLOID DEPOSIT
E37 3 NEUROTOXIC BRAIN DAMAGE
E38 1 NEUROTOXIC BRAIN DAMAGE EVALUATION
E39 1 NEUROTOXIC BRAIN LEVELS
E40 2 NEUROTOXIC CASCADE
E41 1 NEUROTOXIC CELL DEATH
E42 1 NEUROTOXIC CELLS
E43 1 NEUROTOXIC CENTRAL CHOLINERGIC DISRUPTION
E44 2 NEUROTOXIC CHEMICAL
E45 1 NEUROTOXIC CHEMICAL COMBINATIONS
E46 1 NEUROTOXIC CHEMICAL NERVE AGENTS
E47 1 NEUROTOXIC CHEMICAL POTENCY
E48 1 NEUROTOXIC CHEMICAL SUBSTANCES
E49 2 NEUROTOXIC CHEMICAL WARFARE AGENT
E50 1 NEUROTOXIC CHEMICAL-LOADING

E1 1 NEUROTOXIC CHEMICAL-LOADING
E2 9 NEUROTOXIC CHEMICALS
E3 1 NEUROTOXIC CHEMOTHERAPY
E4 1 NEUROTOXIC CHOLINE ANALOGUE
E5 1 NEUROTOXIC COMPLEX
E6 1 NEUROTOXIC COMPLICATION
E7 2 NEUROTOXIC COMPLICATIONS
E8 1 NEUROTOXIC COMPONENT
E9 3 NEUROTOXIC COMPOUND
E10 8 NEUROTOXIC COMPOUNDS
E11 1 NEUROTOXIC CONCENTRATIONS
E12 1 NEUROTOXIC CONDITION
E13 1 NEUROTOXIC CONSEQUENCES
E14 2 NEUROTOXIC CONTAMINATION
E15 1 NEUROTOXIC CONTRIBUTION
E16 1 NEUROTOXIC CONVULSIONS
E17 1 NEUROTOXIC CYANOBACTERIA
E18 1 NEUROTOXIC CYTOKINE
E19 1 NEUROTOXIC CYTOKINES
E20 15 NEUROTOXIC DAMAGE
E21 3 NEUROTOXIC DEGENERATION
E22 1 NEUROTOXIC DEGENERATION PREVENTION
E23 1 NEUROTOXIC DEPLETION
E24 1 NEUROTOXIC DERIVATIVES
E25 1 NEUROTOXIC DESENSITIZING EFFECT
E26 1 NEUROTOXIC DEVELOPMENTAL DEFECTS
E27 3 NEUROTOXIC DISEASE
E28 1 NEUROTOXIC DISEASE SYMPTOMS
E29 1 NEUROTOXIC DISTURBANCE
E30 1 NEUROTOXIC DOSAGE
E31 8 NEUROTOXIC DOSE
E32 3 NEUROTOXIC DOSES
E33 1 NEUROTOXIC DRUGS
E34 1 NEUROTOXIC DYING-BACK PROCESS
E35 2 NEUROTOXIC DYSFUNCTION
E36 77 NEUROTOXIC EFFECT
E37 1 NEUROTOXIC EFFECT ATTENUATION
E38 1 NEUROTOXIC EFFECT DECREASE
E39 1 NEUROTOXIC EFFECT ENHANCEMENT
E40 1 NEUROTOXIC EFFECT MODULATION
E41 1 NEUROTOXIC EFFECTOR
E42 1 NEUROTOXIC EFFECTOR CELLS
E43 180 NEUROTOXIC EFFECTS
E44 2 NEUROTOXIC ENVENOMATION
E45 1 NEUROTOXIC ENVIRONMENTAL EXPOSURES
E46 1 NEUROTOXIC EPILEPTOGENIC IRREVERSIBLE CHOLINES
E47 1 NEUROTOXIC EPITOPE
E48 323 NEUROTOXIC ESTERASE
E49 1 NEUROTOXIC ESTERASE ENZYME
E50 3 NEUROTOXIC ESTERASE INHIBITION

E1 3 NEUROTOXIC ESTERASE INHIBITION
E2 1 NEUROTOXIC ESTERASE INHIBITOR
E3 1 NEUROTOXIC ESTERASE NTE
E4 2 NEUROTOXIC EVENTS
E5 4 NEUROTOXIC EXPOSURE
E6 1 NEUROTOXIC EXPRESSION
E7 1 NEUROTOXIC EXTERASE NTE
E8 5 NEUROTOXIC FACTOR
E9 1 NEUROTOXIC FACTOR SECRETION
E10 7 NEUROTOXIC FACTORS
E11 1 NEUROTOXIC FIBRIL FORMATION
E12 1 NEUROTOXIC FILAMENT-PROMOTING FUNCTION
E13 2 NEUROTOXIC FOOD POISONING
E14 2 NEUROTOXIC FORMS
E15 1 NEUROTOXIC FORNIX LESION
E16 2 NEUROTOXIC FRACTION
E17 3 NEUROTOXIC FRAGMENT
E18 1 NEUROTOXIC FREE RADICAL
E19 2 NEUROTOXIC FREE RADICALS
E20 1 NEUROTOXIC GAIN-OF-FUNCTION
E21 4 NEUROTOXIC HIPPOCAMPAL LESIONS
E22 1 NEUROTOXIC INDICATORS
E23 1 NEUROTOXIC INFLAMMATION
E24 1 NEUROTOXIC INFLAMMATORY RESPONSES
E25 1 NEUROTOXIC INFLUENCE
E26 21 NEUROTOXIC INJURY
E27 1 NEUROTOXIC INJURY TREATMENT
E28 1 NEUROTOXIC INPUT SUSCEPTIBILITY
E29 2 NEUROTOXIC INSECTICIDE
E30 1 NEUROTOXIC INSECTICIDE RECEPTORS
E31 2 NEUROTOXIC INSECTICIDES
E32 7 NEUROTOXIC INSULT
E33 2 NEUROTOXIC INSULTS
E34 2 NEUROTOXIC INTERACTION
E35 1 NEUROTOXIC INTRATHECAL CHEMOTHERAPY
E36 1 NEUROTOXIC ISCHEMIA-RELATED PATHOGENESIS
E37 1 NEUROTOXIC KAINOID AMINO ACID PRECURSORS
E38 1 NEUROTOXIC KAINOID ANALOGS
E39 1 NEUROTOXIC KING COBRA ENVENOMATION
E40 1 NEUROTOXIC KYNURENINE METABOLITE
E41 26 NEUROTOXIC LESION
E42 1 NEUROTOXIC LESION OF DORSAL RAPHE NUCLEUS
E43 3 NEUROTOXIC LESIONING
E44 19 NEUROTOXIC LESIONS
E45 1 NEUROTOXIC LEVELS
E46 1 NEUROTOXIC LOOP
E47 1 NEUROTOXIC LOSS
E48 1 NEUROTOXIC MACROPHAGE PROTEIN
E49 11 NEUROTOXIC MECHANISM
E50 1 NEUROTOXIC MECHANISM OF ACTION

E1 1 NEUROTOXIC MECHANISM OF ACTION
E2 6 NEUROTOXIC MECHANISMS
E3 2 NEUROTOXIC MEDIATOR
E4 1 NEUROTOXIC MEDIATORS
E5 1 NEUROTOXIC MESO-STRIATAL DOPAMINE LESIONS
E6 3 NEUROTOXIC METABOLITE
E7 3 NEUROTOXIC METABOLITES
E8 1 NEUROTOXIC METAL
E9 1 NEUROTOXIC MICROGLIAL-NEURONAL INTERACTIONS
E10 4 NEUROTOXIC MOLECULES
E11 1 NEUROTOXIC MONONUCLEAR PHAGOCYTE FACTORS
E12 1 NEUROTOXIC MULTIPLE INJECTIONS
E13 1 NEUROTOXIC MUSCARINIC AGENT
E14 1 NEUROTOXIC NATURAL PRODUCT
E15 1 NEUROTOXIC NEUROPATHY
E16 1 NEUROTOXIC ORGANIC SOLVENT
E17 1 NEUROTOXIC ORGANIC SOLVENTS
E18 1 NEUROTOXIC ORGANOPHOSPHATE
E19 1 NEUROTOXIC ORGANOPHOSPHATE DRUG
E20 1 NEUROTOXIC ORGANOPHOSPHORUS COMPOUND
E21 1 NEUROTOXIC ORGANOPHOSPHORUS EXPOSURE RISK
E22 6 NEUROTOXIC PEPTIDE
E23 1 NEUROTOXIC PEPTIDE COMPLEMENTARY DNAS
E24 1 NEUROTOXIC PEPTIDE OF PRION PROTEIN PRP106-126
E25 3 NEUROTOXIC PEPTIDES
E26 1 NEUROTOXIC PERIHINAL CORTEX LESIONS
E27 1 NEUROTOXIC PERIRHINAL CORTEX LESION
E28 1 NEUROTOXIC PERIRHINAL CORTEX LESIONS
E29 2 NEUROTOXIC PESTICIDE
E30 4 NEUROTOXIC PESTICIDES
E31 3 NEUROTOXIC PHOSPHOLIPASE
E32 3 NEUROTOXIC PHOSPHOLIPASE A-2
E33 1 NEUROTOXIC PHOSPHOLIPASE A-2 AMMODYTOXIN C

E34 1 NEUROTOXIC PHOSPHOLIPASE A-2 ASSOCIATION
E35 2 NEUROTOXIC PHOSPHOLIPASE A2
E36 1 NEUROTOXIC PHYTODRUGS
E37 1 NEUROTOXIC PIGMENT
E38 1 NEUROTOXIC PLANT PRODUCT INTAKE
E39 1 NEUROTOXIC POISONING
E40 1 NEUROTOXIC POLLUTANTS
E41 1 NEUROTOXIC POLYETHER COMPOUNDS
E42 1 NEUROTOXIC POLYPEPTIDE
E43 2 NEUROTOXIC POLYPEPTIDES
E44 1 NEUROTOXIC POTENCY
E45 24 NEUROTOXIC POTENTIAL
E46 1 NEUROTOXIC PRION PROTEIN PEPTIDE
E47 1 NEUROTOXIC PROCESS
E48 17 NEUROTOXIC PROPERTIES
E49 1 NEUROTOXIC PROTECTION
E50 8 NEUROTOXIC PROTEIN

E1 8 NEUROTOXIC PROTEIN
E2 1 NEUROTOXIC REACTIVE OXYGEN SPECIES
E3 4 NEUROTOXIC REGIMEN
E4 1 NEUROTOXIC REGULATION
E5 3 NEUROTOXIC RESPONSE
E6 2 NEUROTOXIC RESPONSES
E7 1 NEUROTOXIC RHINAL CORTEX LESIONS
E8 1 NEUROTOXIC RHINAL LESION
E9 1 NEUROTOXIC RISKS
E10 1 NEUROTOXIC ROLE
E11 1 NEUROTOXIC ROLES
E12 1 NEUROTOXIC SENSITIVITY
E13 1 NEUROTOXIC SERINE PROTEASE
E14 1 NEUROTOXIC SHELLFISH POISON
E15 26 NEUROTOXIC SHELLFISH POISONING
E16 2 NEUROTOXIC SHELLFISH POISONING NSP
E17 1 NEUROTOXIC SHELLFISH POISONING TOXIN
E18 1 NEUROTOXIC SHELLFISH TOXINS
E19 4 NEUROTOXIC SIDE EFFECT
E20 9 NEUROTOXIC SIDE EFFECTS
E21 1 NEUROTOXIC SIGNALING MOLECULE
E22 1 NEUROTOXIC SIGNALLING RESPONSE
E23 1 NEUROTOXIC SITES
E24 1 NEUROTOXIC SOLUBLE FACTORS
E25 1 NEUROTOXIC SOLVENT WORKERS
E26 2 NEUROTOXIC SOLVENTS
E27 1 NEUROTOXIC SPECIES
E28 1 NEUROTOXIC SPLICE VARIANT
E29 2 NEUROTOXIC STATES
E30 1 NEUROTOXIC STRAINS
E31 1 NEUROTOXIC STRIATAL INJURY
E32 1 NEUROTOXIC STRIATAL LESION
E33 1 NEUROTOXIC STUDIES
E34 1 NEUROTOXIC STUDY
E35 5 NEUROTOXIC SUBSTANCE
E36 1 NEUROTOXIC SUBSTANCE UPTAKE
E37 7 NEUROTOXIC SUBSTANCES
E38 2 NEUROTOXIC SUSCEPTIBILITY
E39 1 NEUROTOXIC SYMPTOM CHECKLIST
E40 1 NEUROTOXIC SYMPTOM QUESTIONNAIRE
E41 5 NEUROTOXIC SYMPTOMS
E42 6 NEUROTOXIC SYNDROME
E43 1 NEUROTOXIC THRESHOLDS
E44 1 NEUROTOXIC TRYPTOPHAN METABOLITE
E45 2 NEUROTOXIC VIRAL PROTEINS
E46 1 NEUROTOXIC VULNERABILITIES
E47 1 NEUROTOXIC VULNERABILITY
E48 1 NEUROTOXIC XENOBIOTICS
E49 1 NEUROTOXIC ZOOCIDE
E50 1 NEUROTOXIC 1-METHYL-4-PHENYL-PYRIDINIUM

E1 1 NEUROTOXIC 1-METHYL-4-PHENYL-PYRIDINIUM
E2 1 NEUROTOXIC-INDUCED CELL DEATH
E3 2 NEUROTOXICA
E4 1 NEUROTOXICACTIVITY
E5 10 NEUROTOXICAL
E6 15 NEUROTOXICALLY
E7 697 NEUROTOXICANT
E8 3 NEUROTOXICANT EXPOSURE
E9 1 NEUROTOXICANT EXPOSURE RATE
E10 1 NEUROTOXICANT METABOLISM
E11 1 NEUROTOXICANT TARGET
E12 1 NEUROTOXICANT TARGET SITES
E13 1 NEUROTOXICANT-INDUCED
E14 1 NEUROTOXICANT-INDUCED BRAIN DAMAGE
E15 1 NEUROTOXICANT-INDUCED DEMYELINATING MODEL
E16 1 NEUROTOXICANT-INDUCED ELEVATION
E17 552 NEUROTOXICANTS
E18 1 NEUROTOXICAS
E19 7 NEUROTOXICATION
E20 7 NEUROTOXICESTERASE
E21 4 NEUROTOXICICITY
E22 20 NEUROTOXICIDAD
E23 2 NEUROTOXICIDADE
E24 1 NEUROTOXICINDUCED
E25 2 NEUROTOXICITA
E26 1 NEUROTOXICITAT
E27 31 NEUROTOXICITE
E28 3 NEUROTOXICITEIT
E29 175 NEUROTOXICITIES
E30 1 NEUROTOXICITIY
E31 28871 NEUROTOXICITY
E32 1 NEUROTOXICITY ACTIVITY
E33 1 NEUROTOXICITY ASSAY
E34 1 NEUROTOXICITY ASSESSMENT
E35 1 NEUROTOXICITY ASSESSMENT TECHNIQUES
E36 1 NEUROTOXICITY ATTENUATION
E37 1 NEUROTOXICITY BIOMARKERS
E38 1 NEUROTOXICITY C
E39 1 NEUROTOXICITY CELLULAR CASCADE
E40 1 NEUROTOXICITY COMPOUND SCREENING
E41 1 NEUROTOXICITY CRITERIA
E42 1 NEUROTOXICITY CURTAILMENT
E43 2 NEUROTOXICITY DATA
E44 1 NEUROTOXICITY DEVELOPMENT
E45 1 NEUROTOXICITY EVALUATION
E46 1 NEUROTOXICITY EXACERBATION
E47 1 NEUROTOXICITY HYPOTHESIS
E48 2 NEUROTOXICITY INDICATOR
E49 1 NEUROTOXICITY INDUCER
E50 1 NEUROTOXICITY INDUCTION ONTOGENY

E1 1 NEUROTOXICITY INDUCTION ONTOGENY
E2 1 NEUROTOXICITY INHIBITION MECHANISMS
E3 1 NEUROTOXICITY LEUKOENCEPHALOMALACIA
E4 1 NEUROTOXICITY MAPPING
E5 6 NEUROTOXICITY MARKER
E6 1 NEUROTOXICITY MARKERS
E7 13 NEUROTOXICITY MECHANISM
E8 9 NEUROTOXICITY MECHANISMS
E9 3 NEUROTOXICITY MEDIATION
E10 3 NEUROTOXICITY MEDIATOR
E11 6 NEUROTOXICITY MEDIATORS
E12 1 NEUROTOXICITY MODEL
E13 1 NEUROTOXICITY MODEL SYSTEMS
E14 1 NEUROTOXICITY MODELS
E15 2 NEUROTOXICITY MODULATION
E16 1 NEUROTOXICITY OPTIMIZATION
E17 1 NEUROTOXICITY POTENTIAL
E18 2 NEUROTOXICITY POTENTIATION
E19 1 NEUROTOXICITY PREDICTION
E20 3 NEUROTOXICITY PREVENTION
E21 1 NEUROTOXICITY PROMOTER
E22 1 NEUROTOXICITY PROPERTIES
E23 2 NEUROTOXICITY PROTECTION
E24 1 NEUROTOXICITY RATING SCALE
E25 1 NEUROTOXICITY REDUCTION
E26 1 NEUROTOXICITY REGULATION
E27 1 NEUROTOXICITY RESEARCH METHOD:BEHAVIORAL TESTI
E28 1 NEUROTOXICITY RESISTANCE
E29 1 NEUROTOXICITY REVERSIBILITY
E30 4 NEUROTOXICITY RISK
E31 3 NEUROTOXICITY RISK ASSESSMENT
E32 3 NEUROTOXICITY RISK ASSESSMENT GUIDELINES
E33 1 NEUROTOXICITY RISK ASSESSMENT PROCESS
E34 1 NEUROTOXICITY RISK FACTORS
E35 1 NEUROTOXICITY ROLE
E36 2 NEUROTOXICITY SCALE
E37 4 NEUROTOXICITY SCREENING
E38 1 NEUROTOXICITY SCREENING BATTERY
E39 1 NEUROTOXICITY SCREENING TESTS
E40 1 NEUROTOXICITY SCREENS
E41 7 NEUROTOXICITY STUDIES
E42 1 NEUROTOXICITY STUDY
E43 2 NEUROTOXICITY SUSCEPTIBILITY
E44 1 NEUROTOXICITY SUSCEPTIBILITY DETECTION
E45 1 NEUROTOXICITY SYMPTOM CHECKLIST-60
E46 0 1 NEUROTOXICITY SYNDROME, LEAD
E47 0 1 NEUROTOXICITY SYNDROME, LEAD, ADULT
E48 0 1 NEUROTOXICITY SYNDROME, LEAD, CHILDHOOD
E49 0 1 NEUROTOXICITY SYNDROME, MANGANESE
E50 0 1 NEUROTOXICITY SYNDROME, MERCURY

E1 0 1 NEUROTOXICITY SYNDROME, MERCURY
E2 414 22 NEUROTOXICITY SYNDROMES
E3 7 NEUROTOXICITY SYNDROMES --BLOOD --BL
E4 4 NEUROTOXICITY SYNDROMES --CLASSIFICATION --CL
E5 9 NEUROTOXICITY SYNDROMES --COMPLICATIONS --CO
E6 76 NEUROTOXICITY SYNDROMES --DIAGNOSIS --DI
E7 20 NEUROTOXICITY SYNDROMES --DRUG THERAPY --DT
E8 2 NEUROTOXICITY SYNDROMES --EMBRYOLOGY --EM
E9 9 NEUROTOXICITY SYNDROMES --ENZYMOLOGY --EN
E10 32 NEUROTOXICITY SYNDROMES --EPIDEMIOLOGY --EP
E11 173 NEUROTOXICITY SYNDROMES --ETIOLOGY --ET
E12 5 NEUROTOXICITY SYNDROMES --GENETICS --GE
E13 39 NEUROTOXICITY SYNDROMES --METABOLISM --ME
E14 3 NEUROTOXICITY SYNDROMES --MICROBIOLOGY --MI
E15 5 NEUROTOXICITY SYNDROMES --MORTALITY --MO
E16 1 NEUROTOXICITY SYNDROMES --NURSING --NU
E17 92 NEUROTOXICITY SYNDROMES --PATHOLOGY --PA
E18 78 NEUROTOXICITY SYNDROMES --PHYSIOPATHOLOGY --PP
E19 29 NEUROTOXICITY SYNDROMES --PREVENTION AND CONTR
E20 65 NEUROTOXICITY SYNDROMES --PSYCHOLOGY --PX
E21 6 NEUROTOXICITY SYNDROMES --RADIONUCLIDE IMAGING
E22 16 NEUROTOXICITY SYNDROMES --THERAPY --TH
E23 1 NEUROTOXICITY SYNDROMES --URINE --UR
E24 2 NEUROTOXICITY SYNDROMES --VETERINARY --VE
E25 1 NEUROTOXICITY TARGET ESTERASE
E26 1 NEUROTOXICITY TARGET TISSUE
E27 3 NEUROTOXICITY TEST
E28 1 NEUROTOXICITY TEST BATTERY
E29 16 NEUROTOXICITY TESTING
E30 2 NEUROTOXICITY TESTING METHOD
E31 1 NEUROTOXICITY THRESHOLD DOSE
E32 1 NEUROTOXICITY TREATMENT
E33 1 NEUROTOXICITY WITH LEUKOENCEPHALOPATHY
E34 1 NEUROTOXICITY-ASSOCIATED OXIDATIVE DAMAGE
E35 1 NEUROTOXICITY-ENHANCING EFFECTS
E36 1 NEUROTOXICITY1
E37 1 NEUROTOXICIY
E38 1 NEUROTOXICKA
E39 1 NEUROTOXICKEHO
E40 2 NEUROTOXICKY
E41 2 NEUROTOXICO
E42 2 NEUROTOXICODYNAMICS

E43 13 NEUROTOXICOL
E44 1 NEUROTOXICOLOGIA
E45 97 NEUROTOXICOLOGIC
E46 1 NEUROTOXICOLOGIC MODEL
E47 1 NEUROTOXICOLOGIC POTENCY
E48 1 NEUROTOXICOLOGIC SEQUELAE
E49 345 NEUROTOXICOLOGICAL
E50 2 NEUROTOXICOLOGICAL ASSESSMENT

E1 2 NEUROTOXICOLOGICAL ASSESSMENT
E2 1 NEUROTOXICOLOGICAL DETECTION
E3 4 NEUROTOXICOLOGICAL EFFECTS
E4 1 NEUROTOXICOLOGICAL ESTIMATION
E5 1 NEUROTOXICOLOGICAL INVESTIGATIONS
E6 1 NEUROTOXICOLOGICAL OUTCOME
E7 1 NEUROTOXICOLOGICAL PROFILE
E8 1 NEUROTOXICOLOGICAL PROPERTIES
E9 1 NEUROTOXICOLOGICAL QUESTIONNAIRE
E10 1 NEUROTOXICOLOGICAL RESEARCH APPLICATION
E11 1 NEUROTOXICOLOGICAL STUDY
E12 1 NEUROTOXICOLOGICAL TESTING METHOD
E13 1 NEUROTOXICOLOGICAL THRESHOLD ASSESSMENT
E14 11 NEUROTOXICOLOGICALLY
E15 1 NEUROTOXICOLOGIE
E16 1 NEUROTOXICOLOGIQUES
E17 4 NEUROTOXICOLOGIST
E18 8 NEUROTOXICOLOGISTS
E19 1093 NEUROTOXICOLOGY
E20 1 NEUROTOXICOLOGY AND TERATOLOGY
E21 1 NEUROTOXICOLOGY FOCUS
E22 1 NEUROTOXICOLOGY GUIDELINES
E23 1 NEUROTOXICOLOGY RESEARCH
E24 1 NEUROTOXICOLOGY RISK ASSESSMENT
E25 1 NEUROTOXICOLOGY STUDIES
E26 1 NEUROTOXICOPATHOLOGY
E27 6 NEUROTOXICOS
E28 14 NEUROTOXICOSES
E29 57 NEUROTOXICOSIS
E30 1 NEUROTOXICQITY
E31 2 NEUROTOXICS
E32 12 NEUROTOXICTY
E33 1 NEUROTOXICY
E34 1 NEUROTOXIDC
E35 1 NEUROTOXIE
E36 3 NEUROTOXIFICATION
E37 26 NEUROTOXIGENIC
E38 5 NEUROTOXIGENICITY
E39 1 NEUROTOXIICITY
E40 1 NEUROTOXIKOLOGIE
E41 3 NEUROTOXIKOLOGISCHE
E42 3 NEUROTOXIKOSE
E43 1 NEUROTOXIKUS
E44 21925 NEUROTOXIN
E45 5 NEUROTOXIN A
E46 2 NEUROTOXIN ACCUMULATION
E47 1 NEUROTOXIN ADMINISTRATION
E48 1 NEUROTOXIN AFFINITY COLUMN CHROMATOGRAPHY

Enter P or PAGE for more
E49 1 NEUROTOXIN ALPHA
E50 1 NEUROTOXIN ALPHA-M-ALPHA-2-3 ANTIBODY COMPLEX

E1 1 NEUROTOXIN ALPHA-M-ALPHA-2-3 ANTIBODY COMPLEX
E2 1 NEUROTOXIN ALPHA-62
E3 1 NEUROTOXIN ANALOGS
E4 1 NEUROTOXIN ANALOGUE
E5 1 NEUROTOXIN ANALYSIS
E6 1 NEUROTOXIN ANTITOXIN
E7 1 NEUROTOXIN APPLICATION
E8 5 NEUROTOXIN ASSOCIATED PROTEIN
E9 1 NEUROTOXIN ASSOCIATED PROTEINS NAPS
E10 1 NEUROTOXIN ASSOCIATION PROTEINS
E11 6 NEUROTOXIN B
E12 1 NEUROTOXIN B GENES
E13 3 NEUROTOXIN B-IV
E14 2 NEUROTOXIN BINDING
E15 1 NEUROTOXIN BINDING DOMAIN
E16 1 NEUROTOXIN BINDING PROTEIN GENE
E17 1 NEUROTOXIN BINDING SITE 2
E18 2 NEUROTOXIN BINDING SITES
E19 1 NEUROTOXIN BIOSYNTHESIS
E20 1 NEUROTOXIN C
E21 1 NEUROTOXIN CANDIDATE
E22 1 NEUROTOXIN CDNA
E23 1 NEUROTOXIN CDNA NEUROTOXIN COMPLEMENTARY DNA
E24 1 NEUROTOXIN CHEMODENERVATION METHOD
E25 1 NEUROTOXIN COMPARISON
E26 1 NEUROTOXIN COMPLEX
E27 1 NEUROTOXIN COMPONENTS
E28 1 NEUROTOXIN CONTENT
E29 1 NEUROTOXIN CONTROL
E30 1 NEUROTOXIN C1
E31 1 NEUROTOXIN D
E32 1 NEUROTOXIN DAMAGE
E33 1 NEUROTOXIN DELTA AMINOLEVULINIC ACID
E34 1 NEUROTOXIN DETECTION
E35 1 NEUROTOXIN DETERMINATION
E36 2 NEUROTOXIN E

S1 67623 NEUROTOX?
S2 939911 INTRACRAN? OR CEREBR? OR HEAD OR CRANI?
S3 7601 S1 AND S2
S4 724856 ENDOCRIN?
S5 479 S3 AND S4
S6 467 RD (unique items)
S7 302824 PITUIT? OR HYPOTHAL?
S8 1908 S1 AND S7
S9 362 S8 AND S4
S10 340 RD (unique items)
S11 284 S10 NOT PY>2000
S12 15509 BOTUL?
S13 293 S12 AND S4
S14 9 S13 AND S2
S15 9 ID (sorted in duplicate order)
S16 321181 S7 OR HYPOPHYS?
S17 31 S16 AND S12
S18 31 ID (sorted in duplicate order)
S19 69771 ACROMEG? OR GIGANT? OR CUSHING? OR HYPERGONAD? OR HYPERTHYROID?
S20 9 S19 AND S12
S21 9 ID (sorted in duplicate order)
S22 57 S19 AND S1
S23 57 ID (sorted in duplicate order)

11/6/1 11892717 99335095 PMID: 10408610
Central serotonin depletion modulates the behavioural, endocrine and physiological responses to repeated social stress and subsequent c-fos expression in the brains of male rats. 1999

11/6/2 (Item 2 from file: 155) 11798149 99237200 PMID: 10220851
Changes in thyroid gland morphology after acute acrylamide exposure. Feb 1999

11/6/3 (Item 3 from file: 155) 11433610 98316256 PMID: 9651548
Inflammatory cytokines: putative regulators of neuronal and neuro- endocrine function. May 1998

11/6/4 (Item 4 from file: 155) 11318034 98197527 PMID: 9536463 Neurochemical and neurobehavioral effects of neonatal administration of beta-N-methylamino-L-alanine and 3,3'-iminodipropionitrile. Mar-Apr 1998

11/6/5 (Item 5 from file: 155) 11081650 97436819 PMID: 9291501
The 139H scrapie agent produces hypothalamic neurotoxicity and pancreatic islet histopathology: electron microscopic studies. 1997

11/6/6 (Item 6 from file: 155) 10489312 96298912 PMID: 8741217
Expression of synaptosomal-associated protein SNAP-25 in endocrine anterior pituitary cells. Apr 1996

11/6/7 (Item 7 from file: 155) 10256185 96057791 PMID: 7549037
Peripheral catecholamines are involved in the neuroendocrine and immune effects of LPS. Jun 1995

11/6/8 (Item 8 from file: 155) 09308632 21065148 PMID: 11133172
Beneficial effect(s) of n-3 fatty acids in cardiovascular diseases: but, why and how? Dec 2000

11/6/9 (Item 9 from file: 155) 09238691 20551243 PMID: 11097794
Mercury, selenium, and cadmium in human autopsy samples from Idrija residents and mercury mine workers. Nov 2000

11/6/10 (Item 10 from file: 155) 08837638 20121712 PMID: 10658625
c-fos expression, behavioural, endocrine and autonomic responses to acute social stress in male rats after chronic restraint: modulation by serotonin. 2000

11/6/11 (Item 11 from file: 155) 08392135 95080114 PMID: 7988433
Hypothalamic - pituitary -adrenal activity during chronic central administration of interleukin-2. Dec 1994

11/6/12 (Item 12 from file: 155) 07665924 93121168 PMID: 1477737
Excitotoxin paraventricular nucleus lesions: stress and endocrine reactivity and oxytocin mRNA levels. Nov 27 1992

11/6/13 (Item 13 from file: 155) 07505454 92369157 PMID: 1324012
Age-related alterations in gonadotropin, adrenocorticotropin and growth hormone secretion. Jun 1992

11/6/14 (Item 14 from file: 155) 07462990 92326534 PMID: 1625526
Influence of amygdala catecholamines on ovarian and adrenal medullary secretion. 1992

11/6/15 (Item 15 from file: 155) 07177648 92039968 PMID: 1718859
Gomori-positive astrocytes: biological properties and implications for neurologic and neuroendocrine disorders. 1991

11/6/16 (Item 16 from file: 155) 06819904 91059846 PMID: 2245800
Neonatal monosodium glutamate abolishes corticotropin-releasing factor-induced epileptogenic activity in rats. Nov-Dec 1990

11/6/17 (Item 17 from file: 155) 05882365 88236495 PMID: 3376130
Changes in plasma corticosterone and catecholamine contents induced by low doses of deltamethrin in rats. May 1988

11/6/18 (Item 18 from file: 155) 05729183 88082584 PMID: 3319562
Pulsatile peptide secretion: encoding of brain messages regulating endocrine and reproductive functions. Nov 1987

11/6/19 (Item 19 from file: 155) 05365545 87043763 PMID: 3775919 Record Identifier: 041848; 00170429

[Male contraception] Mandlig kontraception. Sep 8 1986

11/6/20 (Item 20 from file: 155) 05323325 87001454 PMID: 3756544
Monosodium glutamate neurotoxicity : a sex-specific impairment of blood pressure but not vasopressin in developing rats. Jul 1986

11/6/21 (Item 21 from file: 155) 05268937 86270197 PMID: 3015269
Supersensitive endocrine response to physostigmine in dopamine-depleted rats: a model of depression? Jul 1986

11/6/22 (Item 22 from file: 155) 05078146 86078574 PMID: 3000519
Neurotensin-like immunoreactivity and neurotensin receptors in the rat hypothalamus and in the neurointermediate lobe of the pituitary gland. Dec 9 1985

11/6/23 (Item 23 from file: 155) 04989857 85296956 PMID: 3929189
6-Hydroxydopamine induces degenerative changes in innervation of the rat pituitary gland. Jun 4 1985

11/6/24 (Item 24 from file: 155) 04792502 85098729 PMID: 6097343
Effects of neonatal monosodium glutamate (MSG) treatment on the hormonal and central monoaminergic dynamics associated with acute ether stress in the male rat. Nov 1984

11/6/25 (Item 25 from file: 155) 03750508 82161972 PMID: 7039767
The distribution of enkephalin in the mediobasal hypothalamus of the mouse brain: effects of neonatal administration of MSG. Mar 25 1982

11/6/26 (Item 26 from file: 155) 03724984 82136286 PMID: 6120749
Distribution of somatostatin in the mouse brain: effects of neonatal MSG treatment. Feb 25 1982

11/6/27 (Item 1 from file: 5) 13065019 BIOSIS NO.: 200100272168
Ethylcholine mustard aziridinium ion (AF64A) induces glucocorticoid hypersecretion in rats. 2000

11/6/28 (Item 2 from file: 5) 12937542 BIOSIS NO.: 200100144691
Endangerment of the brain by glucocorticoids: Experimental and clinical evidence. 2000

11/6/29 (Item 3 from file: 5) 12927530 BIOSIS NO.: 200100134679
Immunolocalization of the dopamine transporter in rat pituitary : histochemical and autoradiographic methods. 2000

11/6/30 (Item 4 from file: 5) 12901465 BIOSIS NO.: 200100108614
The role of the amygdala in the pattern of gene expression in the hypothalamic paraventricular nucleus to repeated stress. 2000

11/6/31 (Item 5 from file: 5) 12896955 BIOSIS NO.: 200100104104
Monoamine levels in neonate rats lactationally exposed to 2,4-dichlorophenoxyacetic acid. 2000

11/6/32 (Item 6 from file: 5) 12891145 BIOSIS NO.: 200100098294
Region-specific increase in peripheral-type benzodiazepine receptor binding and neurosteroid formation in Alzheimer's disease. 2000

11/6/33 (Item 7 from file: 5) 12880241 BIOSIS NO.: 200100087390
Neither acute nor chronic exposure to a naturalistic (predator)stressor influences the IL-1beta system, TNF-alpha, TGF-beta1, and neuropeptide mRNAs in specific brain regions. 2000

11/6/34 (Item 8 from file: 5) 12872142 BIOSIS NO.: 200100079291
Molecular mechanism of neurodegeneration induced by Alzheimer's beta-amyloid protein: Channel formation and disruption of calcium homeostasis. 2000

11/6/35 (Item 9 from file: 5) 12680640 BIOSIS NO.: 200000434142
Effects of adrenergic and serotonergic agonists in the amygdala on the hypothalamo - pituitary - adrenocortical axis. 2000

11/6/36 (Item 10 from file: 5) 12680634 BIOSIS NO.: 200000434136
Prolactin and corticosterone secretion in response to acute stress after paraventricular nucleus lesion by ibotenic acid. 2000

11/6/37 (Item 11 from file: 5) 12677249 BIOSIS NO.: 200000430751
Sexual dimorphism in the hypothalamo - pituitary -adrenal (HPA) axis and TNFalpha responses to phospholipase A2-related neurotoxin (from crotalus durissus terrificus) challenge. 2000

11/6/38 (Item 12 from file: 5) 12512229 BIOSIS NO.: 200000265731
Rapid regulated dense-core vesicle exocytosis requires the CAPS protein. 2000

11/6/39 (Item 13 from file: 5) 12438284 BIOSIS NO.: 200000191786
MCS - chemophobia or chemical trauma? 1999

11/6/40 (Item 14 from file: 5) 12410870 BIOSIS NO.: 200000164372
Effect of dioxin on the expression of CYP1A1 in rat brain and pituitary . 1999

11/6/41 (Item 15 from file: 5) 12410148 BIOSIS NO.: 200000163650
Effects of an organophosphate pesticide, quinalphos, on the hypothalamo - pituitary -gonadal axis in adult male rats. 2000

11/6/42 (Item 16 from file: 5) 12369817 BIOSIS NO.: 200000123319
Possible involvement of cytosolic phospholipase A2 in cell death induced by 1-methyl-4-phenylpyridinium ion, a dopaminergic neurotoxin , in GH3 cells. 2000

11/6/43 (Item 17 from file: 5) 12315297 BIOSIS NO.: 200000073164
Effects of estrogen disruptors on the proliferation and the development of immortalized hypothalamic neurons. 1999

11/6/44 (Item 18 from file: 5) 12233969 BIOSIS NO.: 199900528818
Neurotoxin -induced inflammatory response: Modulatory role of endogenous sex steroids. 1999

11/6/45 (Item 19 from file: 5) 12102874 BIOSIS NO.: 199900397723
Androgen-activating enzymes in the central nervous system. 1999

11/6/46 (Item 20 from file: 5) 12079096 BIOSIS NO.: 199900373945
The effects of prenatal stress on the development of hypothalamic paraventricular neurons in fetal rats. 1999

11/6/47 (Item 21 from file: 5) 12043157 BIOSIS NO.: 199900323676
Hyperglycemic effect of a neurotoxic fraction (F3) from Naja haje venom: Role of hypothalamo - pituitary adrenal axis (HPA). 1999

11/6/48 (Item 22 from file: 5) 12027665 BIOSIS NO.: 199900308184
Stressor- or drug-induced sensitization of the corticosterone response is not critically involved in the long-term expression of behavioural sensitization to amphetamine. 1999

11/6/49 (Item 23 from file: 5) 11970084 BIOSIS NO.: 199900223397
Effects of alcohol on the synthesis and expression of hypothalamic peptides. 1999

11/6/50 (Item 24 from file: 5) 11966336 BIOSIS NO.: 199900219649
Hypothalamic involvement in the regulation of maternal behaviour in the rat: Inhibitory roles for the ventromedial hypothalamus and the dorsal/anterior hypothalamic areas. 1999

11/6/51 (Item 25 from file: 5) 11948120 BIOSIS NO.: 199900194229
The Role of the retrochiasmatic area in the control of pineal metabolism. 1999

11/6/52 (Item 26 from file: 5) 11925172 BIOSIS NO.: 199900171281
Toxic effects of cadium on neural estrogen receptors and aromatization of androgens in the male rats during pubertal development. 1999

11/6/53 (Item 27 from file: 5) 11908830 BIOSIS NO.: 199900154939
Responsiveness of the hypothalamo - pituitary -interrenal axis in an amphibian (Bufo terrestris) exposed to coal combustion wastes. 1999

11/6/54 (Item 28 from file: 5) 11866299 BIOSIS NO.: 199900112408
Serotonin transporter (SERT) mRNA and binding site densities in male rat brain affected by sex steroids. 1999

11/6/55 (Item 29 from file: 5) 11838799 BIOSIS NO.: 199900084908
Intracerebral interleukin-1beta impairs response to tumor invasion: Involvement of adrenal catecholamines. 1999

11/6/56 (Item 30 from file: 5) 11828614 BIOSIS NO.: 199900074723
Noradrenergic neurotoxin suppresses gonadotropin-releasing hormone (GnRH) and GnRH receptor gene expression in ovariectomized and steroid-treated rats. 1998

11/6/57 (Item 31 from file: 5) 11788037 BIOSIS NO.: 199900034146
Production of mycotoxins on artificially inoculated building materials. 1998

11/6/58 (Item 32 from file: 5) 11767193 BIOSIS NO.: 199900013302
Stress hormone release and proopiomelanocortin mRNA levels in neonatal rats treated with monosodium glutamate to induce neurotoxic lesions. 1998

11/6/59 (Item 33 from file: 5) 11763002 BIOSIS NO.: 199900009111
Alteration of central serotonin modifies onset and severity of adjuvant-induced arthritis in the rat.1998

11/6/60 (Item 34 from file: 5)11762840 BIOSIS NO.: 199900008949
Synaptophysin immunoreactivity in the rat pituitary : Alternations after 6-hydroxydopamine treatment.1998

11/6/61 (Item 35 from file: 5)11759157 BIOSIS NO.: 199900005266
Neuropeptide Y release in the paraventricular nucleus is decreased during transient hyperphagia induced bdy microinjection of colchicine into the ventromedial nucleus of rats.1998

11/6/62 (Item 36 from file: 5) 11707226 BIOSIS NO.: 199800488957
Decreases in brain glial fibrillary acidic protein (GFAP) are associated with increased serum corticosterone following inhalation exposure to toluene.1998

11/6/63 (Item 37 from file: 5) 11707190 BIOSIS NO.: 199800488921
Food restriction enhances the central rewarding effect of abused drugs. 1998

11/6/64 (Item 38 from file: 5) 11624979 BIOSIS NO.: 199800407282
Ethanol exposure during the last week of gestation in the rat: Inhibition of the prenatal testosterone surge in males without long-term alterations in sex behavior.1998

11/6/65 (Item 39 from file: 5) 11613771 BIOSIS NO.: 199800395541
Involvement of the central noradrenergic system in cholinergic stimulation of the pituitary -adrenal response.1998

11/6/66 (Item 40 from file: 5)11586051 BIOSIS NO.: 199800366747
Alterations in 8-hydroxy-2-(dipropylamino)tetralin-induced neuroendocrine responses after 5,7-dihydroxytryptamine-induced denervation of serotonergic neurons.1998

11/6/67 (Item 41 from file: 5) 11579632 BIOSIS NO.: 199800360328
Neurotoxic lesions induced by monosodium glutamate result in increased adenopituitary proopomelanocortin gene expression and decreased corticosterone clearance in rats.1998

11/6/68 (Item 42 from file: 5)11530670 BIOSIS NO.: 199800312002
Response of the hypothalamo - pituitary -adrenal axis to nicotine.1998

11/6/69 (Item 43 from file: 5)11525971 BIOSIS NO.: 199800307303
Social interactions, brain monoamines, and GABA alterations in MFB-lesioned cats. 1998

11/6/70 (Item 44 from file: 5) 11426964 BIOSIS NO.: 199800208296
Transient expression of the 5alpha-reductase type 2 isozyme in the rat brain in late fetal and early postnatal life. 1998

11/6/71 (Item 45 from file: 5) 11402205 BIOSIS NO.: 199800183537
Interleukin-1 receptor antagonist inhibits transient increase of plasma corticosterone in the phase of trimethyltin-induced hippocampal necrosis. 1998

11/6/72 (Item 46 from file: 5)11358031 BIOSIS NO.: 199800139363
Neuropeptide Y (NPY) Y1 receptor mRNA is upregulated in association with transient hyperphagia and body weight gain: Evidence for a hypothalamic site for concurrent development of leptin resistance. 1998

11/6/73 (Item 47 from file: 5) 11338280 BIOSIS NO.: 199800119612
A phospholipase A2-related snake venom (from Crotalus durissus terrificus) stimulates neuroendocrine and immune functions: Determination of different sites of action. 1998

11/6/74 (Item 48 from file: 5)11276483 BIOSIS NO.: 199800057815
PACAP in visceral afferent nerves supplying the rat digestive and urinary tracts. 1997

11/6/75 (Item 49 from file: 5) 11276210 BIOSIS NO.: 199800057542
Toxicity of the styrene metabolite, phenylglyoxylic acid, *in rats after three months' oral dosing.* 1997

11/6/76 (Item 50 from file: 5)11254742 BIOSIS NO.: 199800036074

Hypothalamic mechanisms mediating glutamate effects on the hypothalamo - pituitary - adrenocortical axis. 1997

11/6/77 (Item 51 from file: 5) 11227873 BIOSIS NO.: 199800009205
Differential 5-HT-mediated regulation of stress-induced activation of proopiomelanocortin (POMC) gene expression in the anterior and intermediate lobe of the pituitary in male rats. 1997

11/6/78 (Item 52 from file: 5) 11227132 BIOSIS NO.: 199800008464
Thyroid hormone-induced plasticity in the adult rat brain. 1997

11/6/79 (Item 53 from file: 5) 11211727 BIOSIS NO.: 199799832872
Glutamatergic stimulation increases the production of reactive oxygen species and lead neurotoxicity in GT1-7 cells.1997

11/6/80 (Item 54 from file: 5) 11209633 BIOSIS NO.: 199799830778
Glucocorticoids and hippocampal neurotoxicity in the aged nonhuman primate.1997

11/6/81 (Item 55 from file: 5) 11206677 BIOSIS NO.: 199799827822
Transient interruption of neural signaling by neurotoxin injection into the ventromedial hypothalamus (VMH) induces temporary hyperphagia and upregulates NPY Y1 receptor gene expression. 1997

11/6/82 (Item 56 from file: 5)11205441 BIOSIS NO.: 199799826586
Brain serotonergic deficits measured in vivo in P-chloroamphetamine rats using novel carbon-11 labeled naphthyl- and isopropenylphenyl-tropane transporter ligands. 1997

11/6/83 (Item 57 from file: 5)11182935 BIOSIS NO.: 199799804080
Serotonin-containing fibers in the suprachiasmatic hypothalamus attenuate light-induced phase delays in mice. 1997

11/6/84 (Item 58 from file: 5) 11150377 BIOSIS NO.: 199799771522
Duration-dependent effects of maternal stress on the development of corticotropin-releasing factor containing neurons in hypothalamic paraventricular nucleus of the fetal rat. 1997

11/6/85 (Item 59 from file: 5) 11141834 BIOSIS NO.: 199799762979
Administration of fenfluramine at different ambient temperatures produces different core temperature and 5-HT neurotoxicity profiles. 1997

11/6/86 (Item 60 from file: 5) 11137040 BIOSIS NO.: 199799758185
Characterization of the effect of Mycoplasma fermentans on the hypothalamo - pituitary -adrenal axis. 1997

11/6/87 (Item 61 from file: 5) 11137012 BIOSIS NO.: 199799758157
Recovery of hypothalamic NMDA-induced c-fos expression following neonatal glutamate (MSG) lesions. 1997

11/6/88 (Item 62 from file: 5) 11130480 BIOSIS NO.: 199799751625
Botulinum neurotoxin F, a VAMP-specific endopeptidase, inhibits Ca-2+-stimulated GH secretion from rat pituitary cells. 1997

11/6/89 (Item 63 from file: 5) 11122207 BIOSIS NO.: 199799743352
Time-dependent effects of repeated amphetamine treatment on norepinephrine in the hypothalamus and hippocampus assessed with in vivo microdialysis. 1997

11/6/90 (Item 64 from file: 5) 11107164 BIOSIS NO.: 199799728309
Ion channel formation of Alzheimer's beta amyloid protein and neurodegeneration. 1997

11/6/91 (Item 65 from file: 5) 11089533 BIOSIS NO.: 199799710678
Distribution of the 5-hydroxytryptamine-2C receptor protein in adult rat brain and spinal cord determined using a receptor-directed antibody: Effect of 5,7-dihydroxytryptamine. 1997

11/6/92 (Item 66 from file: 5) 11079181 BIOSIS NO.: 199799700326
Waglerin-1 modulates gamma-aminobutyric acid activated current of murine hypothalamic neurons. 1997

11/6/93 (Item 67 from file: 5) 11060585 BIOSIS NO.: 199799681730
Circadian rhythms in hypothalamus are altering under the influence of xenobiotics. 1997

11/6/94 (Item 68 from file: 5)11055724 BIOSIS NO.: 199799676869
Inhibition of luteinizing hormone (LH) release by lead in a rat lifetime exposure model: Effects via the opiate system. 1997

11/6/95 (Item 69 from file: 5) 11053928 BIOSIS NO.: 199799675073
Opposite effects of ethanol and stress on orphanin FQ gene expression in the anterior hypothalamus . 1997

11/6/96 (Item 70 from file: 5) 11030798 BIOSIS NO.: 199799651943
The role of cAMP in ethanol-regulated beta-endorphin release from hypothalamic neurons. 1997

11/6/97 (Item 71 from file: 5) 11026720 BIOSIS NO.: 199799647865
Neurotoxic consequences of central long-term administration of interleukin-2 in rats. 1997

11/6/98 (Item 72 from file: 5) 11018042 BIOSIS NO.: 199799639187
Alzheimer's disease amyloid beta-protein forms Zn-2+-sensitive, cation-selective channels across excised membrane patches from hypothalamic neurons. 1997

11/6/99 (Item 73 from file: 5) 11011461 BIOSIS NO.: 199799632606
Electroporation of botulinum neurotoxin serotypes A and B into GH-3 cells: Substrate cleavage and growth hormone secretion. 1996

11/6/100 (Item 74 from file: 5) 10986131 BIOSIS NO.: 199799607276
Pituitary adenylate cyclase activating peptide (PACAP) in salivary glands of the rat: Origin, and secretory and vascular effects. 1997

11/6/101 (Item 75 from file: 5) 10982764 BIOSIS NO.: 199799603909
Interleukin-6 affects scopolamine-induced amnesia, but not brain amino acid levels in mice. 1997

11/6/102 (Item 76 from file: 5)10981839 BIOSIS NO.: 199799602984
Pathogenesis of the neurotoxicity caused by anti-GD2 antibody therapy. 1997

11/6/103 (Item 77 from file: 5) 10978320 BIOSIS NO.: 199799599465
The epidemiology of estrogen replacement therapy and Alzheimer's disease. 1997

11/6/104 (Item 78 from file: 5) 10947814 BIOSIS NO.: 199799568959
Expression of nitric oxide synthase in hypothalamic nuclei following axonal injury or colchicine treatment. 1997

11/6/105 (Item 79 from file: 5) 10937438 BIOSIS NO.: 199799558583
Nephrogenic diabetes insipidus in a lethargic lithium-treated patient. 1997

11/6/106 (Item 80 from file: 5) 10914372 BIOSIS NO.: 199799535517
Attenuation by pituitary adenylate cyclase activating peptide in glutamate-induced toxicity of cultured retinal neurons. 1997

11/6/107 (Item 81 from file: 5) 10905116 BIOSIS NO.: 199799526261
Combined phentermine/fenfluramine administration enhances depletion of serotonin from central terminal fields. 1997

11/6/108 (Item 82 from file: 5) 10905091 BIOSIS NO.: 199799526236
Dietary preferences in monosodium glutamate-lesioned rats: Age-variable influence of hypothalamic neuropeptide Y. 1997

11/6/109 (Item 83 from file: 5) 10905041 BIOSIS NO.: 199799526186
Repeated cocaine administration down-regulates glucocorticoid receptors in the rat brain cortex and hippocampus. 1996

11/6/110 (Item 84 from file: 5) 10904953 BIOSIS NO.: 199799526098
Dopamine depletion in the rostral nucleus accumbens alters the cerebral metabolic response to cocaine in the rat. 1997

11/6/111 (Item 85 from file: 5) 10904756 BIOSIS NO.: 199799525901
Long-term exposure to ozone alters peripheral and central catecholamine activity in rats. 1997

11/6/112 (Item 86 from file: 5) 10900385 BIOSIS NO.: 199799521530
Quantitation of dopamine transporter mRNA in the rat brain: Mapping, effects of "binge" cocaine administration and withdrawal. 1997

11/6/113 (Item 87 from file: 5) 10895355 BIOSIS NO.: 199799516500
Involvement of SNAP-25 in TRH-induced exocytosis in pituitary GH4C1 cells. 1997

11/6/114 (Item 88 from file: 5) 10889946 BIOSIS NO.: 199799511091
Peripheral markers of neurochemical effects among styrene-exposed workers. 1996

11/6/115 (Item 89 from file: 5) 10885701 BIOSIS NO.: 199799506846
Evidence showing that beta-endorphin regulates cyclic guanosine 3',5'-monophosphate (cGMP) efflux: Anatomical and functional support for an interaction between opiates and nitric oxide. 1997

11/6/116 (Item 90 from file: 5) 10876996 BIOSIS NO.: 199799498141
Behavioral and biochemical alterations in median and dorsal raphe nuclei lesioned cats. 1997

11/6/117 (Item 91 from file: 5) 10843968 BIOSIS NO.: 199799465113
Thyrotropin-releasing hormone (TRH) is markedly increased in the rat brain following soman-induced convulsions. 1997

11/6/118 (Item 92 from file: 5) 10840718 BIOSIS NO.: 199799461863
Overflow of noradrenaline and dopamine in frontal cortex after (N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine) (DSP-4) treatment: In vivo microdialysis study in anaesthetized rats. 1997

11/6/119 (Item 93 from file: 5) 10838470 BIOSIS NO.: 199799459615 The tuberomammillary nucleus projections in the control of learning, memory and reinforcement processes: Evidence for an inhibitory role. 1997

11/6/120 (Item 94 from file: 5) 10792098 BIOSIS NO.: 199799413243
Low-affinity sulfonylurea binding sites reside on neuronal cell bodies in the brain. 1997

11/6/121 (Item 95 from file: 5) 10791791 BIOSIS NO.: 199799412936
The effect of estradiol-induced hypothalamic pathology on sulfated glycoprotein-2 (clusterin) expression in the hypothalamus . 1997

11/6/122 (Item 96 from file: 5) 10746990 BIOSIS NO.: 199799368135
An immunotoxin, anti-VIP antibody-ricin A chain conjugate eliminates neurons in the hypothalamic suprachiasmatic nucleus selectively and abolishes the circadian rhythm of water intake. 1996

11/6/123 (Item 97 from file: 5) 10717241 BIOSIS NO.: 199799338386
Pituitary adenylate cyclase activating polypeptide (PACAP38) protects cultured cerebral and cerebellar cortical neurons against glutamate neurotoxicity in rats. 1996

11/6/124 (Item 98 from file: 5) 10673226 BIOSIS NO.: 199799294371
Immunocytochemical localization of seleno-glutathione peroxidase in the adult mouse brain. 1996

11/6/125 (Item 99 from file: 5) 10673073 BIOSIS NO.: 199799294218
Consumption of a high dietary dose of monosodium glutamate fails to affect extracellular glutamate levels in the hypothalamic arcuate nucleus of adult rats. 1996

11/6/126 (Item 100 from file: 5) 10649585 BIOSIS NO.: 199699270730
Neonatal cocaine exposure effects on the volume of the sexually dimorphic nucleus of the preoptic area. 1996

11/6/127 (Item 101 from file: 5) 10649580 BIOSIS NO.: 199699270725
Effect of prenatal cocaine on foot shock-induced CRF mRNA and hnRNA in the paraventri nucleus of hypothalamus (PVN). 1996

11/6/128 (Item 102 from file: 5) 10649571 BIOSIS NO.: 199699270716
Periadolescent cocaine and stress effects on reproductive maturation and estrous cyclicity in the female rat. 1996

11/6/129 (Item 103 from file: 5) 10642099 BIOSIS NO.: 199699263244
Effect of 5,7-dihydroxytryptamine on audiogenic seizures in genetically epilepsy-prone rats. 1996

11/6/130 (Item 104 from file: 5) 10629006 BIOSIS NO.: 199699250151
Interaction between norepinephrine and prostaglandin E-2 in the preoptic area of guinea pigs. 1996

11/6/131 (Item 105 from file: 5) 10596783 BIOSIS NO.: 199699217928
Neurotoxin -induced transient hyperphagia is negatively correlated with hypothalamic neuropeptide Y. 1996

11/6/132 (Item 106 from file: 5) 10591351 BIOSIS NO.: 199699212496
Induction of IL-1b expression and cell death by hexachlorobenzene in differentiated N1E-115 neuroblastoma. 1996

11/6/133 (Item 107 from file: 5) 10590429 BIOSIS NO.: 199699211574
Effects of DSP-4 on distribution of dopamine-beta-hydroxylase immunoreactivity (DBHir) in male zebra finch brains. 1996

11/6/134 (Item 108 from file: 5) 10568271 BIOSIS NO.: 199699189416
Behavioral, neurochemical, and neuromorphological effects of deltamethrin in adult rats. 1996

11/6/135 (Item 109 from file: 5) 10540160 BIOSIS NO.: 199699161305
No changes in behaviour, nigro-striatal system neurochemistry or neuronal cell death following toxic multiple oral paraquat administration to rats. 1996

11/6/136 (Item 110 from file: 5) 10484930 BIOSIS NO.: 199699106075
A selective role of brainstem noradrenergic neurons in oxytocin release from the neurohypophysis following noxious stimuli in the rat. 1996

11/6/137 (Item 111 from file: 5) 10428218 BIOSIS NO.: 199699049363
Kainic acid versus carbachol induced emotional-defensive response in the cat. 1996

11/6/138 (Item 112 from file: 5) 10388802 BIOSIS NO.: 199699009947
Forebrain pathway mediating stress-induced renin secretion. 1996

11/6/139 (Item 113 from file: 5) 10388198 BIOSIS NO.: 199699009343
Loss of vasopressin-immunoreactive neurons in alcoholics is dose-related and time-dependent. 1996

11/6/140 (Item 114 from file: 5) 10373041 BIOSIS NO.: 199698827959
Energy and glutamate dependency of 3-nitropropionic acid neurotoxicity in culture. 1996

11/6/141 (Item 115 from file: 5) 10330761 BIOSIS NO.: 199698785679
Norepinephrine depletion impairs neuroendocrine responses to fear but not novel environmental stimuli in the rat. 1996

11/6/142 (Item 116 from file: 5) 10300382 BIOSIS NO.: 199698755300
Persistent neuroendocrine effects of neonatal glutamic acid (GLU). 1995

11/6/143 (Item 117 from file: 5)10292415 BIOSIS NO.: 199698747333
Effects of neonatal administration of octreotide, a long-lasting somatostatin analogue, on growth hormone regulation in the adult rat. 1996

11/6/144 (Item 118 from file: 5) 10281874 BIOSIS NO.: 199698736792
Methylenedioxymethamphetamine-induced serotonin deficits are followed by partial recovery over a 52-week period: Part II: Radioligand binding and autoradiography studies. 1996

11/6/145 (Item 119 from file: 5) 10244307 BIOSIS NO.: 199698699225
Interleukin-1-beta: Common cause of Alzheimer's disease and diabetes mellitus. 1995

11/6/146 (Item 120 from file: 5) 10241427 BIOSIS NO.: 199698696345
The increase in growth hormone secretion in experimentally induced arthritic rats is an adaptive process involved in the regulation of inflammation. 1996

11/6/147 (Item 121 from file: 5) 10233790 BIOSIS NO.: 199698688708
Enhanced feeding response to neuropeptide Y in hypothalamic neuropeptide Y-depleted rats. 1996

11/6/148 (Item 122 from file: 5) 10229298 BIOSIS NO.: 199698684216
Effect of domoic acid on brain amino acid levels. 1995

11/6/149 (Item 123 from file: 5) 10228561 BIOSIS NO.: 199698683479
Role of calcium in brain aging. 1995

11/6/150 (Item 124 from file: 5) 10178323 BIOSIS NO.: 199698633241
Lesions of the afferent catecholaminergic pathways inhibit the temporal activation of the CRH and POMC gene expression and ACTH release induced by human interleukin-1-beta in the male rat. 1995

11/6/151 (Item 125 from file: 5)10162822 BIOSIS NO.: 199698617740
Effects of prenatal stressful stimuli on serotonin content in the hypothalamus and pituitary - adrenal response to conditioned fear stress in adult offspring.BOOK TITLE: International Congress Series; Serotonin in the central nervous system and periphery 1995

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1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP), and gamma-vinyl-gamma-aminobutyric acid (gamma-vinyl GABA) alter neurotransmitter concentrations in the nervous tissue of the goldfish (Carassius auratus) but not the cockroach (Periplaneta americana). 1993

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A PET radiotracer for studying serotonin uptake sites: Carbon-11 McN-5652Z. 1993

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Vitamin E protects hypothalamic beta-endorphin neurons from estradiol neurotoxicity . 1992

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Changes in cholecystokinin receptor binding in rat brain after selective damage of locus coeruleus projections by DSP-4 treatment. 1992

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Dopamine catabolism in goldfish (Carassius auratus) brain and pituitary : Lack of influence of catecholestrogens on dopamine catabolism and gonadotropin secretion. 1992

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Effects of neonatal monosodium glutamate on peptide mRNA expression in rat endocrine and visual CNS structures. 1992

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5,7-DHT infusions into the medial hypothalamus facilitate lordosis in preweanling rats. 1992

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Hippocampal glucocorticoid and mineralocorticoid receptor gene expression is unaltered in Alzheimer's disease. 1992

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In-Vitro Modulation Of Beta Endorphin Secretion From Rat Neurointermediate Pituitary After 6 Hydroxydopamine-Induced Degeneration Of Nerve Terminals 1990

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The Effects Of Lesions In The Lateral Septal And Hippocampal Areas On The Humoral Immune Response Of Adult Female Rats 1987

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Neurochemical Lesion Of The Nucleus Locus Coeruleus Increases Neophobia In A Specific Exploration Task But Doe Not Modify Endocrine Response To Moderate Stress 1988

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Lead Age And Aggression In Male Mice 1986

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Effects Of Neonatal Monosodium Glutamate Treatment On The Hormonal And Central Monoaminergic Dynamics Associated With Acute Ether Stress In The Male Rat 1984

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Effect Of D L-Alpha Aminoadipate On The Mediobasal Hypothalamus And Endocrine Function In The Rat 1984

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Effect Of Norepinephrine On The Pituitary Adreno Corticotropic Activation By Ether Stress The In-Vitro Release Of Acth By The Adeno Hypophysis Of Male And Female New Born Rats 1984

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Brain Part Mono Amines In The Neuro Endocrine Mechanisms Activated By Immobilization Stress In The Rat 1984

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Distribution Of Somatostatin In The Mouse Brain Effects Of Neo Natal Mono Sodium Glutamate Treatment 1982

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11/6/282 (Item 256 From File: 5) 02969637 Biosis No.: 000069077755
Acute Central Stimulation Of Luteinizing Hormone By Parenterally Administered N Methyl-D L Aspartic-Acid In The Male Rat 1980

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Acute Elevations Of Serum Luteinizing Hormone Induced By Kainic-Acid N Methyl Aspartic-Acid Or Homo Cysteic-Acid 1978

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Analysis Of The Disruption In Hypothalamic Pituitary Regulation In Rats Treated Neo Natally With Mono Sodium L Glutamate Evidence For The Involvement Of Tubero Infundibular Cholinergic And Dopaminergic Systems In Neuro Endocrine Regulation 1977

11/7/11 (Item 11 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
08392135 95080114 PMID: 7988433
Hypothalamic - pituitary -adrenal activity during chronic central administration of interleukin-2.
Hanisch U K; Rowe W; Sharma S; Meaney M J; Quirion R
Douglas Hospital Research Center, Department of Psychiatry, Montreal, Quebec, Canada.
Endocrinology (UNITED STATES) Dec 1994, 135 (6) p2465-72, ISSN 0013-7227 Journal Code: 0375040
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
The cytokine interleukin-2 (IL-2) exerts numerous effects within the immune as well as the central nervous system and is thought to serve as a humoral signal in their communication. Brain-derived or blood-borne IL-2 may also control the activity of the hypothalamic - pituitary -adrenal (HPA) axis at various levels of regulation. In this study we investigated whether persistently elevated levels of central IL-2, which are associated with several diseases or induced during immunotherapeutic use of this cytokine, could induce long term activation of the HPA axis. Adult male Sprague-Dawley rats received an intracerebroventricular infusion of the recombinant cytokine at a rate of 5 U/h (equivalent to 2.5 ng/h or 162 fmol/h) by means of osmotic minipumps. Control animals received heat-inactivated IL-2. After 7 days of continuous infusion, blood samples were taken at intervals of 4 h over a period of 24 h, and plasma levels of ACTH and corticosterone (CORT) were determined. IL-2 caused a significant increase in ACTH levels during the later portion of the dark phase of the cycle. Plasma CORT concentrations were significantly elevated over almost the whole diurnal cycle. Measurements of CORT-binding globulin concentrations revealed IL-2-induced decreases during the dark phase, resulting in a marked increase in free CORT. Additionally, after 11 days of chronic infusion both groups of animals underwent a 20-min restraint stress. IL-2-treated animals showed stress-induced increases in plasma ACTH and CORT that were not significantly different from those of animals treated with heat-inactivated IL-2. Along with the alteration of HPA activity seen in the IL-2-treated animals, chronic delivery of the cytokine caused periventricular tissue damage and gliosis. Taken together, the data reflect the capacity of IL-2 to modulate neuroendocrine activity over an extended period of treatment. Moreover, the IL-2-induced effects on HPA activity seen here may help to explain some of the endocrine disturbances seen in patients undergoing IL-2 immunotherapy. Record Date Created: 19950106 Record Date Completed: 19950106

11/7/12 (Item 12 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
07665924 93121168 PMID: 1477737
Excitotoxin paraventricular nucleus lesions: stress and endocrine reactivity and oxytocin mRNA levels.
Callahan M F; Thore C R; Sundberg D K; Gruber K A; O'Steen K; Morris M
Department of Medicine, Bowman Gray School of Medicine, Wake Forest University Medical Center, Winston-Salem, NC 27157.
Brain research (NETHERLANDS) Nov 27 1992, 597 (1) p8-15, ISSN 0006-8993 Journal Code: 0045503
Contract/Grant No.: HL35112; HL; NHLBI; RR05404; RR; NCRR Document type: Journal Article Languages: ENGLISH
Main Citation Owner: NLM Record type: Completed

Electrolytic lesion of the paraventricular nucleus (PVN) of the hypothalamus blocks the tachycardia response to stress. The current study examined the effects of chemical lesion of PVN parvocellular neurons on the cardiovascular and endocrine responses to stress and on the content of hypothalamic oxytocin (OT) mRNA levels. Acute footshock stress increased heart rate in both ibotenic acid lesion and control groups of animals; however, the tachycardia was significantly lower in animals with a PVN lesion than the controls. Lesion of the PVN also attenuated the increase in plasma OT induced by stress, 4-fold in the lesion group versus 20-fold for the controls. There was not a generalized decrease in hormonal responsiveness since the OT response to an osmotic challenge was exaggerated in the lesion group. There was no difference between the groups in the arterial pressure and vasopressin responses to acute stress. Neurotoxin lesions of the PVN also resulted in significant depletions of VP and OT in all levels of the spinal cord and decreased OT levels in the dorsal brainstem. Ibotenic acid lesions of the PVN resulted in no significant changes in OT mRNA in the PVN, SON and PP. In addition, the 48-h dehydration resulted in a significant increase in plasma OT and OT mRNA in the PVN. These data indicate that the parvocellular neurons of the PVN play a role in integration of cardiovascular and endocrine responses to both stressful and osmotic stimuli and provide further evidence that parvocellular OT and VP neurons project to the brainstem and spinal cord. Record Date Created: 19930209 Record Date Completed: 19930209

11/7/17 (Item 17 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
05882365 88236495 PMID: 3376130
Changes in plasma corticosterone and catecholamine contents induced by low doses of deltamethrin in rats.
de Boer S F; van der Gugten J; Slangen J L; Hijzen T H
Department of Psychophysiology, State University of Utrecht, The Netherlands.
Toxicology (NETHERLANDS) May 1988, 49 (2-3) p263-70, ISSN 0300-483X Journal Code: 0361055
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
The effects of low doses of (S)-alpha-cyano-3-phenoxybenzyl (1R)-cis-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropanecarboxylate (Roussel UCLAF, Paris, France), (deltamethrin) upon sympathetic-adrenomedullary and pituitary -adrenocortical activity were investigated in rats by measuring plasma noradrenaline (NA), adrenaline (A) and corticosterone (CS) concentrations. Blood was sampled from freely-moving animals provided with heart catheters at short intervals up to 60 min after intravenous administration of deltamethrin (0.05, 0.15 and 0.45 mg/kg) or vehicle. Behavioral activity was recorded shortly after the sampling times. Time course and magnitude of the biochemical changes were compared with the effects of exposure to uncontrollable white noise in a similar sampling and recording procedure. Dose-dependent increases were observed for NA and A as well as for CS contents. The dose-response relations however, were different among the neuro-edocrine respondents. Discrete step-wise increases were observed for plasma CS only, indicating greater sensitivity for neurotoxical actions. Already at a dose of 0.15 mg/kg of deltamethrin, CS contents rose to values that were considerably higher than those found during noise exposure. In contrast, plasma CA concentrations increased to noise stress values only after the 0.45 mg/kg dose. The behavioral activity pattern appeared to resemble both CA patterns. The results suggest that rather low doses of deltamethrin elicit vigorous autonomic and neuro- endocrine responses that indicate high levels of stress, presumably caused by the neurotoxic effect of the insecticide. Record Date Created: 19880628 Record Date Completed: 19880628

11/7/19 (Item 19 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
05365545 87043763 PMID: 3775919 Record Identifier: 041848; 00170429
[Male contraception] Mandlig kontraception.
Kjaergaard N
Ugeskrift for laeger (DENMARK) Sep 8 1986, 148 (37) p2335-8, ISSN 0041-5782 Journal Code: 0141730
TJ: UGESKRIFT FOR LAEGER. Document type: Journal Article ; English Abstract Languages: DANISH
Main Citation Owner: NLM Other Citation Owner: PIP; POP Abstract Source: PIP Record type: Completed
Male contraceptive drugs meeting the criteria of efficacy, easy applicability and reversibility -- and also having limited side effects -- are discussed. These drugs affect the hypothalamus , the hypophysis, the ductus deferens, and the testes. Gestagens inhibit the secretion of follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Gonadotropic hormones inhibit spermatogenesis by producing intratesticular concentration of testosterone. Recently, a combination of gestagen and androgen has been used to avoid decreased libido. Oral medroxyprogesterone acetate and percutaneous testosterone produce azoospermia, but with serious side effects, i.e., hirsutism and acne; however, they can be reduced by iv administration. Cyproterone acetate inhibits androgen secretion, and a daily dosage of 5-10 mg slightly reduces the sperm count; however, higher doses can lead to thromboembolitic effects. Combining cyproterone with an androgen prevents negative effects on the libido. Buserelin administered iv or as a nasal spray stimulates the secretion of LH and FSH from the hypophysis 20-40 times more effectively than does natural LH release of the hormone (LH-RH). Gossypol exerts its inhibitory effect on the epithelium of testes, but can cause hypokalemia. It has been used successfully in a Chinese experiment involving 9000 men who received daily oral doses of 20 mg for 60-70 days. Cytostatic agents also produce azoospermia, but are not recommended. Implantation of a copper wire into the ductus deferens has been tried in animal experiments with mixed results. Vasectomy has been performed on 50 million men since the 1960's (48,000 in 1981 in Denmark). It has to be regarded as a partially irreversible operation. The oral administration of the adrenergic blocking agent, phenoxybenzamine, at the rate of 20 mg per day has produced azoospermia; however, more research is needed to understand its side effects. Finally, chlorohydrin is not usable because of its neurotoxic and nephrotoxic effects. Record Date Created: 19861128 Record Date Completed: 19861128

11/7/23 (Item 23 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
04989857 85296956 PMID: 3929189
6-Hydroxydopamine induces degenerative changes in innervation of the rat pituitary gland.
Saland L C; Comunas F
Neuroscience letters (NETHERLANDS) Jun 4 1985, 57 (1) p49-55, ISSN 0304-3940 Journal Code: 7600130 Contract/Grant No.: R-8005; PHS; RR08139; RR; NCRR Document type: Journal Article Languages: ENGLISH
Main Citation Owner: NLM Record type: Completed
Administration of 6-hydroxydopamine to adult male rats by peripheral injections induces degenerative changes in nerve terminals innervating cells of the pituitary intermediate lobe. Additional animals were treated with 5-hydroxydopamine, which produced images of nerve profiles containing vesicles with electron-dense centers, indicative of uptake of the false catecholamine (CA) neurotransmitter. Endocrine cells showed cytologic evidence of activation of synthetic and secretory compartments. The ultrastructural observations suggest that innervation to opiomelanocortin cells is sensitive to a CA-specific neurotoxin and strengthens the hypothesis that CA-containing fibers play a role in intermediate lobe neuroregulation. Record Date Created: 19851003 Record Date Completed: 19851003

11/7/27 (Item 1 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
13065019 BIOSIS NO.: 200100272168
Ethylcholine mustard aziridinium ion (AF64A) induces glucocorticoid hypersecretion in rats.
AUTHOR: Endo Yutaka(a)
AUTHOR ADDRESS: (a)Department of Physiology, School of Medicine, University of Occupational and Environmental Health, Kitakyushu**Japan
JOURNAL: Neuroscience Research Supplement (24):pS166 2000 MEDIUM: print CONFERENCE/MEETING: 23rd Annual Meeting of the Japan Neuroscience Society and the 10th Annual Meeting of the Japanese Neural Network Society Yokoha Japan September 04-06, 2000 ISSN: 0921-8696 RECORD TYPE: Citation LANGUAGE: English SUMMARY LANGUAGE: English

11/7/35 (Item 9 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
12680640 BIOSIS NO.: 200000434142
Effects of adrenergic and serotonergic agonists in the amygdala on the hypothalamo - pituitary -adrenocortical axis.
AUTHOR: Feldman Shaul(a); Newman Michael E; Weidenfeld Joseph
AUTHOR ADDRESS: (a)Department of Neurology, Hadassah University Hospital, Jerusalem**Israel
JOURNAL: Brain Research Bulletin 52 (6):p531-536 August, 2000 MEDIUM: print ISSN: 0361-9230
DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English SUMMARY LANGUAGE: English
ABSTRACT: The effect of direct administration of adrenergic and serotonergic (5-HT) agonists into the central nucleus of the amygdala (AMG) on the hypothalamo - pituitary -adrenal (HPA) axis have been studied in intact male rats and in animals with 6-hydroxydopamine (6-OHDA) or 5,7-dihydroxytryptamine (5,7-DHT) neurotoxic lesions in the paraventricular nucleus of the hypothalamus (PVN). In intact animals, the administration of phenylephrine, an alpha1 adrenergic agonist or 8-hydroxy-2-(di-n-propylamino)-tetralin (8-OH-DPAT) a 5-HT1A agonist caused depletion of median eminence corticotropin releasing hormone and a rise in serum adrenocorticotrophic hormone (ACTH) and corticosterone (CS) levels. Isoproterenol a beta agonist was more effective than phenylephrine and a 5-HT1B agonist CP-93, 129 was less effective than 8-OH-DPAT on the adrenocortical activity. The 6-OHDA or 5,7-DHT hypothalamic lesions prevented the stimulatory effects of phenylephrine and 8-OH-DPAT, respectively, which where injected into the AMG, on serum ACTH and CS levels. In view of our previous studies on the effects of the adrenergic and 5-HT antagonists in the AMG and the present data, it is suggested that norepinephrine and 5-HT play an important role in the stimulatory effect of the AMG on the HPA axis. These effects depend on the presence of these excitat neurotransmitters in the PVN.

11/7/40 (Item 14 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
12410870 BIOSIS NO.: 200000164372
Effect of dioxin on the expression of CYP1A1 in rat brain and pituitary .
AUTHOR: Huang P(a); Rannug A(a); Ahlbom E(a); Hakansson H(a); DiBona G; Ceccatelli S(a)
AUTHOR ADDRESS: (a)Institute of Environmental Medicine, Karolinska Institutet, S-171 77, Stockholm**Sweden
JOURNAL: Society for Neuroscience Abstracts. 25 (1-2):p1826 1999 CONFERENCE/MEETING: 29th Annual Meeting of the Society for Neuroscience. Miami Beach, Florida, USA October 23-28, 1999 SPONSOR: Society for Neuroscience ISSN: 0190-5295 RECORD TYPE: Citation LANGUAGE: English SUMMARY LANGUAGE: English

11/7/41 (Item 15 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
12410148 BIOSIS NO.: 200000163650
Effects of an organophosphate pesticide, quinalphos, on the hypothalamo - pituitary -gonadal axis in adult male rats.
AUTHOR: Sarkar R; Mohanakumar K P(a); Chowdhury M
AUTHOR ADDRESS: (a)Indian Institute of Chemical Biology, 4, Raja SC Mullick Road, Calcutta, 700 032**India
JOURNAL: Journal of Reproduction and Fertility. 118 (1):p29-38 Jan., 2000 ISSN: 0022-4251 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English SUMMARY LANGUAGE: English

ABSTRACT: The effects of chronic sub-lethal doses (7-14 mg kg-1 a day for 15 days) of quinalphos were evaluated in adult male rats for changes in testicular morphology, circulatory concentrations of hormones (LH, FSH, prolactin and testosterone), activities of acetylcholinesterase (AChE) and angiotensin converting enzyme (ACE) as well as metabolism of biogenic amines (dopamine, noradrenaline and 5-hydroxytryptamine (5-HT)) in the hypothalamus and pituitary . Hormones were assayed by radioimmunoassay or chemiluminescent immunoassay (testosterone). The enzymes were estimated after spectrophotometry and the biogenic amines by HPLC-electrochemistry. Sub-lethal chronic administration of quinalphos resulted in: decreased testicular mass and AChE activity in central as well as peripheral organs; increased serum LH, FSH, prolactin and testosterone concentrations; decreased pituitary or increased testicular ACE activity; severe disruption of spermatogenesis with increasing doses of pesticide; and no significant effects on dopamine, noradrenaline or 5-HT concentrations in the hypothalamus or pituitary . Administration of oestradiol (50 mug per rat a day) during pesticide treatment resulted in: a significant decrease in the mass of the testis and accessory sex organs; decreases in serum LH, FSH, testosterone concentrations; an increase in prolactin concentration; and a decrease in dopamine or an increase in noradrenaline and 5-HT in the hypothalamus or pituitary . Oestradiol had a marked effect: in pesticide-treated animals, the pesticide effects were significantly reversed. This indicates that in pesticide toxicity, the hypothalamo - pituitary -gonadal axis is operational. Since many of the observed pesticide effects could be inhibited by oestradiol, it is suggested that the pesticide acts directly on the gonadotrophins. In conclusion, quinalphos decreases fertility in adult male rats by affecting the pituitary gonadotrophins.

11/7/44 (Item 18 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
12233969 BIOSIS NO.: 199900528818
Neurotoxin -induced inflammatory response: Modulatory role of endogenous sex steroids.
AUTHOR: Chisari A N(a); Gaillard R C; Giovambattista A(a); Voirol M-J; Spinedi E(a)
AUTHOR ADDRESS: (a)Neuroendocrine Unit, IMBICE, La Plata**Argentina
JOURNAL: Neuroimmunomodulation 6 (6):p465 Nov.-Dec., 1999 CONFERENCE/MEETING: 4th International Congress of the International Society for Neuroimmunomodulation Lugano, Switzerland September 29-October 2, 1999
SPONSOR: International Society for Neuroimmunomodulation ISSN: 1021-7401 RECORD TYPE: Citation LANGUAGE: English

11/7/47 (Item 21 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
12043157 BIOSIS NO.: 199900323676
Hyperglycemic effect of a neurotoxic fraction (F3) from Naja haje venom: Role of hypothalamo - pituitary adrenal axis (HPA).
AUTHOR: El-Fiky Maha A(a)
AUTHOR ADDRESS: (a)Zoology Department, Faculty of Science, Ain Shams University, Cairo**Egypt
JOURNAL: Journal of Natural Toxins 8 (2):p203-212 June, 1999 ISSN: 1058-8108 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English SUMMARY LANGUAGE: English
ABSTRACT: The effect of bolus intravenous injection of sub-LD50 (35 mug Kg-1) of the neurotoxic fraction (F3) of the Egyptian cobra Naja haje on the plasma level of ACTH and serum levels of cortisol, insulin, glucose, total lipids, triacylglycerols, free fatty acids, total cholesterol, HDL and LDL-cholesterol, and glycogen content of liver and kidneys were studied in rabbit pretreated with cyproteron acetate (CA) or saline solution and propylene glycol (PG) to elucidate the possible role of the hypothalamo - pituitary adrenal (HPA) axis in the venom fraction-induced hyperglycemia. F3 increased cortisol and insulin level in both groups, whereas ACTH was found to decrease subsequent to the treatment. Serum glucose level was elevated by F3 treatment and this effect was substantiated in CA-treated rabbits. This hyperglycemia was concomitant with a decline in glycogen content of the liver and kidneys. A decline in serum total lipids, triacylglycerols, and free fatty acids was observed following F3 treatment, and this effect was intensified by CA-pretreatment. These data suggest that F3 stimulates glucocorticoid release from adrenocortical cells which, in turn, may modulate both insulin and glucose turnover to maintain hyperglycemia during stress period. The possible underlying mechanisms were discussed.

11/7/56 (Item 30 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11828614 BIOSIS NO.: 199900074723
Noradrenergic neurotoxin suppresses gonadotropin-releasing hormone (GnRH) and GnRH receptor gene expression in ovariectomized and steroid-treated rats.
AUTHOR: Kang S S; Son G H; Seong J Y; Choi D; Kwon H B; Lee C C; Kim K(a)
AUTHOR ADDRESS: (a)Dep. Mol. Biol., Coll. Nat. Sci., Seoul Natl. Univ., Seoul 151-742**South Korea
JOURNAL: Journal of Neuroendocrinology 10 (12):p911-918 Dec., 1998 ISSN: 0953-8194 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The present study was designed to investigate whether noradrenergic neurotransmission regulates the gene expression of gonadotropin-releasing hormone (GnRH) in the preoptic area and GnRH receptor in the pituitary . To this end, N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine (DSP4, 50 mg/kg), an intraperitoneal (i.p.) injection of selective noradrenergic neurotoxin , was administered 1 h before progesterone (1 mg) treatment in ovariectomized and estradiol-treated prepubertal rats. Treatment with DSP4 effectively blocked the progesterone-induced increase in hypothalamic noradrenaline content, but not dopamine content, indicating that DSP4 selectively inhibits noradrenergic neurotransmission. DSP4 significantly blocked progesterone-induced increase in serum luteinizing hormone (LH) concentrations as well as GnRH release from hypothalamic fragments incubated in vitro. DSP4 concomitantly down-regulated GnRH mRNA levels in the preoptic area, as determined by competitive reverse transcription-polymerase chain reaction. DSP4 also clearly down-regulated progesterone-induced GnRH receptor mRNA levels in the pituitary , whereas it failed to alter LHbeta mRNA levels. In summary, blockade of noradrenergic neurotransmission with DSP4 resulted in profound reductions of hypothalamic GnRH and pituitary GnRH receptor gene expression.

11/7/60 (Item 34 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11762840 BIOSIS NO.: 199900008949
Synaptophysin immunoreactivity in the rat pituitary : Alternations after 6-hydroxydopamine treatment.
AUTHOR: Saland Linda C(a); Thomas Deidre; Morales Marti; Gaddy Jasmine
AUTHOR ADDRESS: (a)Dep. Neurosciences, Univ. New Mexico School Medicine, Basic Med. Sci. Building, Albuqurque, NM 8**Mexico
JOURNAL: Endocrine 9 (2):p201-206 Oct., 1998 ISSN: 1355-008X DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Synaptophysin (SN) is a synaptic-vesicle-associated membrane
protein whose presence is indicative of intact, functional synapses. This study examines the presence of SN in pituitary gland innervation after neurotoxin -induced denervation followed by reinnervation. Immunostaining of rat pituitary neurointermediate lobe tissue for SN reveals a pattern of dot-like densities in the intermediate lobe and intensely stained dispersed regions in the neural lobe of normal animals. In rats treated with 6-hydroxydopamine (6-OHDA), a catecholamine neurotoxin , by peripheral injection, there is a significant depletion of the SN immunostaining in the intermediate lobe, as well as a significant reduction of SN immunoreactivity in the neural lobe, in animals studied 1 wk after drug treatment, with computer analysis of the tissue sections. At 3 wk after 6-OHDA, there is a partial recovery of immunoreactivity for SN in the neural lobe in many tissue sections, and the intermediate lobe also contains only relatively sparse staining for the synaptic protein. Computer analysis revealed that at 3 wk after 6-OHDA, both lobes still had reduced SN immunoreactivity, but the difference in levels measured did not achieve statistical significance. These results contrast with the prior finding of significant recovery of immunoreactivity for GAP-43, a growth and regeneration-associated protein, in intermediate lobe innervation of rats treated with the same drug regimen. We suggest that 6-OHDA treatment damages synaptic vesicle integrity in both the intermediate and neural lobes of the pituitary , and that recovery is in progress, but not complete at 3 wk after the drug is administered.

11/7/61 (Item 35 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11759157 BIOSIS NO.: 199900005266
Neuropeptide Y release in the paraventricular nucleus is decreased during transient hyperphagia induced bdy microinjection of colchicine into the ventromedial nucleus of rats.
AUTHOR: Jain Mukul R; Dube Michael G; Kalra Satya P; Kalra Pushpa S(a)
AUTHOR ADDRESS: (a)Dep. Physiol., Univ. Fla., Coll. Med., P.O. Box 100274, Gainesville, FL 32610-0274**USA
JOURNAL: Neuroscience Letters 256 (1):p21-24 Oct. 30, 1998 ISSN: 0304-3940 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Disruption of neural signaling in the ventromedial nucleus (VMN) of rats by microinjection of the neurotoxin colchicine (COL) results in transient hyperphagia accompanied by enhanced weight gain. We tested the hypothesis that release of neuropeptide Y (NPY), a potent orexigenic signal is augmented within the paraventricular nucleus (PVN) of COL-treated hyperphagic rats. Adult male rats were microinjected bilaterally with either COL (4 mug/0.5 mul in saline) or saline in the VMN and a push-pull guide cannula aimed at the PVN was implanted for analysis of extra-cellular NPY. COL-injected rats gained 37.8 +- 6.1 g while the saline-injected rats lost 9.3 +- 3.4 g during the 4 days following surgery. On day 4, post-injection, the PVN of these rats was perfused with artificial cerebrospinal fluid via the push-pull cannula. NPY levels in perfusates collected at 10 min intervals from hyperphagic, COL-injected rats were markedly diminished. Cumulative NPY efflux over the 180 min sampling period was significantly less in COL-treated (27.7 +- 6.0 pg) versus saline-injected control rats (I 10.6 +- 32.2 pg; P < 0.05). These results show that impairment of neural signaling in the VMN by COL suppressed NPY release in the PVN. These observations taken together with previous studies showing diminution in preproNPY mRNA in the arcuate nucleus (ARC) and NPY levels in the PVN are in accordance with the thesis that the VMN normally exerts a facilitatory influence on NPYergic signaling in the ARC-PVN axis.

11/7/66 (Item 40 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11586051 BIOSIS NO.: 199800366747
Alterations in 8-hydroxy-2-(dipropylamino)tetralin-induced neuroendocrine responses after 5,7-dihydroxytryptamine-induced denervation of serotonergic neurons.
AUTHOR: Van De Kar Louis(a); Li Qian; Cabrera Theresa M; Brownfield Mark S; Battaglia George
AUTHOR ADDRESS: (a)Dep. Pharmacol., Stricth Sch. Med., Loyola Univ. Chicago, 2160 S. First Ave., Maywood, IL 60153**USA
JOURNAL: Journal of Pharmacology and Experimental Therapeutics 286 (1):p 256-262 July, 1998 ISSN: 0022-3565
DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: In the present study, we examined denervation-induced changes in the sensitivity of hypothalamic postsynaptic serotonin1A (5-HT1A) receptor function with respect to changes in the dose-dependent elevation in plasma hormones (adrenocorticotropic hormone (ACTH), corticosterone, prolactin, oxytocin, prolactin, renin and vasopressin) by the 5-HT1A agonist 8-hydroxy-2-(dipropylamino)tetralin (8-OH-DPAT). Rats received intracerebroventricular (i.c.v.) injections of the serotonin

neurotoxin 5,7-dihydroxytryptamine (5,7-DHT) or vehicle (0.1% ascorbate in saline) 3 weeks before challenge with increasing doses of 8-OH-DPAT (0, 10, 50 or 200 mug/kg s.c.). The effectiveness of 5,7-DHT-induced destruction of serotonergic neurons was confirmed by a 93% reduction in (3H)paroxetine-labeled 5-HT uptake sites in the hypothalamus . No changes in basal levels of ACTH, corticosterone, oxytocin, prolactin, renin and vasopressin were observed in rats that received i.c.v. 5,7-DHT injections. The dose-response curves for 8-OH-DPAT-induced elevations of plasma corticosterone and prolactin levels were shifted to the left in rats treated with 5,7-DHT, whereas no significant difference in the ACTH dose-response curve was observed between rats treated with vehicle and rats treated with 5,7-DHT. In contrast, the maximal oxytocin response to 8-OH-DPAT was attenuated in rats treated with 5,7-DHT. A 5,7-DHT-induced decline in the synthesis of oxytocin could explain this phenomenon. Although 8-OH-DPAT did not increase plasma levels of renin or vasopressin in rats treated with vehicle, 8-OH-DPAT produced an elevation (75%) in plasma renin concentration but not in vasopressin levels in rats that received i.c.v. injections of 5,7-DHT. No change was observed in (3H)8-OH-DPAT labeled 5-HT1A receptors in the hypothalamus . In summary, denervation of hypothalamic serotonergic nerve terminals produces supersensitivity of some neuroendocrine responses to 8-OH-DPAT independent of changes in the density of hypothalamic 5-HT1A receptors.

11/7/68 (Item 42 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11530670 BIOSIS NO.: 199800312002
Response of the hypothalamo - pituitary -adrenal axis to nicotine.
AUTHOR: Matta Shannon G(a); Fu Yitong; Valentine James D; Sharp Burt M
AUTHOR ADDRESS: (a)Endocrine Neurosci. Lab., MSC-D3, Minneapolis Med. Res. Foundation, 914 South Eight St., Minneap**USA
JOURNAL: Psychoneuroendocrinology 23 (2):p103-113 Feb., 1998 ISSN: 0306-4530 DOCUMENT TYPE: Literature Review RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Nicotine has been shown to be a potent stimulus for the secretion of the stress-responsive hormones, adrenocorticotropin (ACTH) and prolactin. This paper reviews the findings by our laboratory and others that demonstrate the polysynaptic pathways involved in the neuroendocrine responses to systemic nicotine. It will focus primarily on the hypothalamo - pituitary -adrenal (HPA) axis and the effect of nicotine on ACTH secretion, with supplementary information on prolactin secretion, where relevant. Data are presented demonstrating that nicotine acts via a central mechanism to stimulate indirectly the release of ACTH from the anterior pituitary corticotropes. Nicotine does not appear to act directly at the hypothalamic paraventricular nucleus (PVN), the site of the corticotropin-releasing hormone (CRH) neurons crucial to the regulation of ACTH. However, brainstem catecholaminergic regions projecting to the PVN showed a regionally selective and dose-dependent sensitivity to nicotine, particularly the noradrenergic/adrenergic nucleus tractus solitarius (NTS). A reduction in the modulatory effect of these catecholamines (by neurotoxic lesion, synthetic enzyme inhibitors or adrenergic receptor antagonists) resulted in an inhibition of nicotine-stimulated ACTH secretion. In addition, blockade of nicotinic cholinergic receptors (NAchRs) in the brainstem by the antagonist, mecamylamine, resulted in a dose-dependent reduction in norepinephrine (NE) release from terminals in the PVN, and a concomitant reduction in plasma ACTH. The differential sensitivity of these receptors to the nicotinic agonists, cytisine and nicotine, reflects the heterogeneity of the NAchR subtypes involved. The desensitization characteristics of the neuroendocrine responses to both acute and chronic nicotine exposure are indicative of an alteration in these NAchRs.

11/7/76 (Item 50 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11254742 BIOSIS NO.: 199800036074
Hypothalamic mechanisms mediating glutamate effects on the hypothalamo - pituitary -adrenocortical axis.
AUTHOR: Feldman S(a); Weidenfeld J
AUTHOR ADDRESS: (a)Dep. Neurol., Hadassah Univ. Hosp., PO Box 12000, Jerusalem 91120**Israel
JOURNAL: Journal of Neural Transmission 104 (6-7):p633-642 1997 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The effect of local administration of glutamate into the hypothalamic paraventricular nucleus (PVN) on the hypothalamo - pituitary adrenocortical (HPA) axis was studied in male rats. Glutamate caused CRH-41 depletion from the median eminence (ME) and a consequent rise in ACTH and corticosterone (CS) serum levels. In rats pretreated with systemic dexamethasone (dex) these effects were completely inhibited. The administration of the glucocorticoid receptor antagonist RU-38486 abolished the inhibitory effect of dex on the adrenocortical discharge. In addition, the depletion of hypothalamic nore-pinephrine (NE) and serotonin (5-HT) by specific neurotoxins administered into the ventral noradrenergic blundle or into the raphe nuclei respectively, inhibited the response of serum ACTH and CS following PVN glutamate administration. These data indicate that glutamate stimulated the HPA axis via the release of ME CRH-41 into the portal circulation. This response is steroid sensitive involving type II glucocorticoid receptors. Hypothalamic NE and 5-HT participate in the glutamate induced HPA axis activation.

11/7/77 (Item 51 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11227873 BIOSIS NO.: 199800009205
Differential 5-HT-mediated regulation of stress-induced activation of proopiomelanocortin (POMC) gene expression in the anterior and intermediate lobe of the pituitary in male rats.
AUTHOR: Garcia-Garcia Luis; Fuentes Jose A(a); Manzanares Jorge
AUTHOR ADDRESS: (a)Inst. Pluridisciplinar, Univ. Complutense, Paseo Juan XXIII 1, 28040 Madrid**Spain
JOURNAL: Brain Research 772 (1-2):p115-120 Oct. 24, 1997 ISSN: 0006-8993 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The purpose of the present study was to examine the role of 5-hydroxytryptamine (5-HT) neurons in mediating the effects of stress on proopiomelanocortin (POMC) gene expression in the anterior and intermediate lobes of the pituitary gland. To this aim, the effects of 5-HT depletion induced by administration of the neurotoxin 5,7-dihydroxytryptamine (5,7-DHT; 200 mug/rat; i.c.v.; 7 days) were investigated on POMC mRNA levels in the anterior and intermediate lobe of control and restraint-stressed rats. Three hours after brief exposure to diethyl ether (2 min) followed by 60 min of restraint stress increased POMC mRNA levels in the anterior and intermediate lobe of the pituitary . 5,7-DHT neurotoxic lesion, which resulted in a marked depletion of 5-HT (below the level of sensitivity of the neurochemical assay, 6 pg/sample) but not of dopamine or norepinephrine concentrations in the periventricular nucleus of the hypothalamus , had no effect on basal POMC mRNA levels in the anterior or intermediate lobe of the pituitary . However, 5-HT depletion further increased POMC mRNA levels in the anterior pituitary and completely blocked POMC mRNA level enhancement induced in the intermediate lobe of stressed rats. These results suggest a possible inhibitory 5-HT tone on POMC gene expression in the anterior pituitary and a stimulatory 5-HT tone in the intermediate lobe of the pituitary under these experimental conditions of stress. It appears, therefore, that 5-HT exerts a differential regulation of stress-induced activation of POMC gene expression in the anterior and intermediate lobes of the pituitary in male rats.

11/7/81 (Item 55 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11206677 BIOSIS NO.: 199799827822
Transient interruption of neural signaling by neurotoxin injection into the ventromedial hypothalamus (VMH) induces temporary hyperphagia and upregulates NPY Y1 receptor gene expression.
AUTHOR: Kalra S P(a); Dube M G; Xu B; Farmerie W G; Kalra P S
AUTHOR ADDRESS: (a)Dep. Neurosci., Univ. Fla. Coll. Med., Gainesville, FL 32610**USA
JOURNAL: Society for Neuroscience Abstracts 23 (1-2):p1344 1997 CONFERENCE/MEETING: 27th Annual Meeting of the Society for Neuroscience New Orleans, Louisiana, USA October 25-30, 1997 ISSN: 0190-5295 RECORD TYPE: Citation LANGUAGE: English

11/7/85 (Item 59 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11141834 BIOSIS NO.: 199799762979
Administration of fenfluramine at different ambient temperatures produces different core temperature and 5-HT neurotoxicity profiles.
AUTHOR: Malberg Jessica E; Seiden Lewis S(a)
AUTHOR ADDRESS: (a)Dep. Pharmacological Physiological Sciences, Univ. Chicago, 947 E. 58th St., Chicago, IL 60637**USA
JOURNAL: Brain Research 765 (1):p101-107 1997 ISSN: 0006-8993 RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: This study investigated the effect of two different ambient temperatures on fenfluramine-induced 5-HT neurotoxicity , Fenfluramine (FEN) (12.5 mg/kg times 4; injections made hourly) or saline (SAL) was administered to rats in either a normal laboratory temperature of 24 degree C or a warm environment of 30 degree C. Animals were kept at that ambient temperature for 20 h after FEN administration. Ambient temperature was controlled to +- 0.5 degree C and rat core temperature was continually measured using a non-invasive apparatus. FEN-treated rats at 24 degree C displayed a core temperature hypothermia with a peak low of 33.8 degree C, and this core temperature hypothermia lasted for 20 h after FEN administration. Rats treated with FEN at 30 degree C displayed a significant core temperature hyperthermia for 4 h after the first drug injection compared to SAL-treated groups, with a peak core temperature of 38.6 degree C. 2 weeks after FEN injections, brain regions were analyzed by HPLC. Both groups of FEN-treated rats showed decreases in 5-HT and 5-HIAA in the hippocampus, frontal cortex, somatosensory cortex, striatum, hypothalamus and septum. However, FEN rats treated at 30 degree C had significantly greater decreases (26-35%) in 5-HT compared to FEN-treated rats at 24 degree C in the frontal cortex, hippocampus, striatum and somatosensory cortex and significantly greater decreases (26-50%) in 5-HIAA in the frontal cortex, hippocampus and somatosensory cortex. This study indicates fenfluramine can produce neurotoxicity in rats that display either a core temperature hypothermia or hyperthermia, although hyperthermic rats have greater 5-HT and 5-HIAA depletions than the hypothermic rats.

11/7/94 (Item 68 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11055724 BIOSIS NO.: 199799676869
Inhibition of luteinizing hormone (LH) release by lead in a rat lifetime exposure model: Effects via the opiate system.
AUTHOR: Ronis M J J(a); Parker J; Shaw M; Rector C; Lehigh M; Badger T M
AUTHOR ADDRESS: (a)Arkansas Children's Hosp. Res. Inst., Univ., Arkansas Med. Sci., Little Rock, AR**USA
JOURNAL: Biology of Reproduction 56 (SUPPL. 1):p100 1997 CONFERENCE/MEETING: Thirtieth Annual Meeting of the Society for the Study of Reproduction Portland, Oregon, USA August 2-5, 1997 ISSN: 0006-3363 RECORD TYPE: Citation LANGUAGE: English

11/7/118 (Item 92 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10840718 BIOSIS NO.: 199799461863
Overflow of noradrenaline and dopamine in frontal cortex after (N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine) (DSP-4) treatment: In vivo microdialysis study in anaesthetized rats.
AUTHOR: Kask Ants(a); Harro Jaanus; Tuomainen Paivi; Rago Lembit; Mannisto Pekka T

AUTHOR ADDRESS: (a)Dep. Pharmacol. Univ. Tartu, Ulikooli 18, EE-2400 Tartu **Estonia
JOURNAL: Naunyn-Schmiedeberg's Archives of Pharmacology 355 (2):p267-272 1997 ISSN: 0028-1298
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The changes in the extracellular concentration of endogenous noradrenaline and dopamine in the frontal cortex following pretreatment with noradrenergic neurotoxin DSP-4 (N-(2-chloroethyl)-N-ethyl-2-bromobenzylamine) were studied by in vivo microdialysis in rats anaesthetized with chloral hydrate. Noradrenaline and dopamine levels in frontal cortex were detected only when the uptake inhibitor, nomifensine (10 mu-M) was present in dialysis fluid. Under those conditions, the Na+ channel agonist veratridine and a depolarising concentration of potassium chloride (60 mM), applied locally through the microdialysis probe, increased the overflow of noradrenaline. Tetrodotoxin had an opposite effect. These results indicate that most of the noradrenaline probably arose from exocytotic release. Noradrenaline efflux in the frontal cortex of DSP-4 pretreated rats (52+-6.1 fmol/sample) did not differ significantly from that of the control animals (69+-4.9 fmol/sample). Dopamine efflux was not changed either (64+-9.6 and 62+-3.9 fmol/sample, respectively). The a-2-adrenoceptor antagonist, atipamezole (3 mg/kg i.p.), increased the overflow of noradrenaline in the frontal cortex of saline-treated rats by 100%, whereas in DSP-4 treated rats the increase was only around 30%. The overflow of dopamine was not changed under the conditions described. The effect of atipamezole in DSP-4 treated rats may be of smaller magnitude due to the diminished pool of releasable noradrenaline or due to a downregulation of presynaptic alpha-2-adrenoceptors in the frontal cortex. The perfusion of 60 mM KCl at the end of the experiment unexpectedly produced equivalent increases in noradrenaline and dopamine content in dialysates of both vehicle and DSP-4 treated rats. We conclude that the uptake inhibitor, nomifensine, and atipamezole, which had a stronger effect in vehicle-treated animals, reduced the effect of KCl-stimulation and masked the true difference in changes of noradrenaline efflux. Post-mortem tissue concentrations of noradrenaline 7 days after DSP-4 administration (50 mg/kg) were significantly reduced in the frontal cortex (54%), hippocampus (62.5%) and to lesser extent in the hypothalamus (27%) as compared to vehicle-treated rats. Dopamine and 3,4-dihydroxyphenylacetic acid concentrations were not changed confirming the efficacy and selectivity of the DSP-4 lesion. These results demonstrate that one week after DSP-4 treatment the extracellular levels of noradrenaline and dopamine as assessed by in vivo microdialysis are not changed in the frontal cortex, but atipamezole-stimulated release of noradrenaline is decreased in DSP-4 treated rats.

11/7/117 (Item 91 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10843968 BIOSIS NO.: 199799465113
Thyrotropin-releasing hormone (TRH) is markedly increased in the rat brain following soman-induced convulsions.
AUTHOR: Kubek Michael J(a); Shih Tsung Ming; Meyerhoff James L
AUTHOR ADDRESS: (a)J. D. VanNuys Medical Sci. Build., Dep. Anat., Indiana Univ. Sch. Med., 635 Barnhill Dr., Indian**USA
JOURNAL: Brain Research 747 (2):p328-331 1997 ISSN: 0006-8993 RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Soman is an organophosphorus (OP) compound which irreversibly inhibits acetylcholinesterase (AChE), the primary synaptic inactivator of acetylcholine. Resultant excessive cholinergic activity elicits generalized convulsions and brain lesions. Recent evidence suggests that other neurotransmitter/neuromodulator systems may be affected by the OP compounds as well. Since we have shown that both electrically and chemically induced seizures cause significant and prolonged increases in the neuropeptide thyrotropin-releasing hormone (TRH) in epileptogenic sites, we examined soman-induced convulsion effects on CNS TRH. Rats were injected with either soman (100 mu-g/kg SC; equivalent to 0.9 LD-50) or saline and observed for convulsive activity. Forty-eight hours post injection, dramatic increases of TRH over control levels were seen in frontal cortex (30-fold), pooled cortex (24-fold), hippocampus (16-fold), piriform cortex (14-fold), entorhinal cortex (11-fold), and amygdala (2-fold). No change was observed in either hypothalamus or pituitary . Our results demonstrate, for the first time, a substantial effect of an OP on a specific neuropeptide system in vivo. The neurochemical and behavioral consequences of the soman-induced increases in TRH, especially in the frontal cortex, are presently unknown. Clearly, much more work is required to discern the exact role TRH has following soman exposure.

11/7/122 (Item 96 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10746990 BIOSIS NO.: 199799368135
An immunotoxin, anti-VIP antibody-ricin A chain conjugate eliminates neurons in the hypothalamic suprachiasmatic nucleus selectively and abolishes the circadian rhythm of water intake.
AUTHOR: Shimizu K(a); Nagai K; Nakagawa H
AUTHOR ADDRESS: (a)Div. Protein Metab., Inst. Protein Res., Osaka Univ., 3-2 Yamado-Oka, Suita, Osaka 565**Japan
JOURNAL: Brain Research Bulletin 41 (6):p369-378 1996 ISSN: 0361-9230 RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: In mammals, a master circadian oscillator is known to be located in the suprachiasmatic nucleus (SCN) of the hypothalamus . We examined the function of SCN neurons involved in the mechanism of circadian rhythm of water intake by lesioning them with an immunotoxin, anti-vasoactive intestinal polypeptide (VIP) antibody-ricin A conjugate. We found that the immunotoxin had a specific lethal effect on cultured PC12h cells when VIP was added to the medium. When the conjugate was infused into the third cerebral ventricle of rats above the SCN, two specific types of selective lesions of neurons were observed in the SCN: selective lesions of neurons containing arginine vasopressin (AVP) (AVP-neurons), and selective lesions of neurons containing VIP (VIP neurons). The former lesions caused disappearance of the circadian rhythm of drinking behavior, whereas the latter lesions did not affect the rhythm of water intake under constant dim lighting. Lesions that did not selectively affect one of these neurochemically identified SCN cell populations were also observed after the infusion of the conjugate or normal rabbit serum immunoglobulin G-ricin A chain conjugate. If these nonspecific lesions included entire region of the SCN, the circadian rhythm of water intake was abolished. These findings suggest that SCN neurons bearing VIP receptors such as AVP neurons, but not VIP neurons, may be involved in the mechanism of the circadian rhythm of water intake.

11/7/131 (Item 105 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10596783 BIOSIS NO.: 199699217928
Neurotoxin -induced transient hyperphagia is negatively correlated with hypothalamic neuropeptide Y.
AUTHOR: Dube M G(a); Xu B; Kalra S P; Kalra P S
AUTHOR ADDRESS: (a)Dep. Physiol., Univ. Florida College Med., Gainesville, FL 32610**USA
JOURNAL: Society for Neuroscience Abstracts 22 (1-3):p466 1996 CONFERENCE/MEETING: 26th Annual Meeting of the Society for Neuroscience Washington, D.C., USA November 16-21, 1996 ISSN: 0190-5295 RECORD TYPE: Citation LANGUAGE: English

11/7/143 (Item 117 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10292415 BIOSIS NO.: 199698747333
Effects of neonatal administration of octreotide, a long-lasting somatostatin analogue, on growth hormone regulation in the adult rat.
AUTHOR: Slama Annie(a); Bluet-Pajot Marie-Therese; Mounier Francoise; Videau Catherine; Kordon Claude; Epelbaum Jacques
AUTHOR ADDRESS: (a)INSERM, Unite 159, 2ter, rue d'Alesia, F-75014 Paris** France
JOURNAL: Neuroendocrinology 63 (2):p173-180 1996 ISSN: 0028-3835 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The pulsatile pattern of GH secretion slowly develop in the postnatal period concomitantly with the dual network GHRH and somatostatin (SRIH) hypothalamic neurons. We investigated whether an early postnatal treatment with a long ac SRIH analogue, octreotide, could affect maturation and subsequent operation of those networks in the adult rat. Octreotide administration (5 mu-g/rat SC) every other day during the first 10 days of life resulted in growth retardation in the adult. In parallel, the amplitude of plasma GH secretory episodes in free moving unanesthetized animals was markedly reduced. The numbers of arcuate GHRH mRNA-containing and periventricular SRIH-mRNA containing neurons were not affected by the treatment. GHRH mRNA levels per neuron however was decreased by 30%, and median eminence GHRH stores by 50%. SRIH expression in the arcuate nucleus was also diminished, as was the number of 125I-SRIH labeled neurons in that nucleus. The effects of octreotide were compared to the hyposomatotropinemia induced by administration of monosodium glutamate (MSG), every other day during the first 10 days of life. Growth retardation and inhibition of GH secretory episodes in adult rats neonatally treated with MSG were slightly more pronounced than after octreotide. In contrast to octreotide, MSG induced a massive loss of GHRH neurons and a concomitant decrease in 125I-SRIH binding. Somatostatin did not protect GHRH neurons against the neurotoxic action of MSG since octreotide treatment did not further affect any of the parameters impaired by MSG. In conclusion, these experiments demonstrate that neonatally injected octreotide cannot counteract the toxic effect of MSG on arcuate neurons. However, a neonatal treatment with the SRIH agonist affects permanently growth rate and GH pulsatility. This effect is mediated in the hypothalamus by permanently impairing the neural networks that control GH secretion.

11/7/145 (Item 119 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10244307 BIOSIS NO.: 199698699225
Interleukin-1-beta: Common cause of Alzheimer's disease and diabetes mellitus.
AUTHOR: Holden R J(a); Mooney P A
AUTHOR ADDRESS: (a)Med. Res. Unit, Univ. Wollongong, Northfields Ave., Wollongong, NSW 2522**Australia
JOURNAL: Medical Hypotheses 45 (6):p559-571 1995 ISSN: 0306-9877 DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Alzheimer disease is characterized by the presence of beta-amyloid protein deposits, neurofibrillary tangles and cholinergic dysfunction throughout the hippocampal region. in addition, the hippocampus, hypothalamus and olfactory bulb - the three areas where the insulin receptors are most dense - are also subject to neurodegeneration. The exact cause of the beta-amyloid deposits and NFTs is unknown. However, it is our intention to explicate the various pathogenic pathways through which Alzheimer disease arises. Fundamentally, the structural and metabolic damage found in Alzheimer disease is due to sustained elevation of interleukin-1-beta, a feature which is also found in insulin-dependent diabetes mellitus. Similarly, the beta-AP deposits found in the Alzheimer brain share the same molecular structure as the amylin deposits found in the pancreatic beta-cells in non-insulin-dependent diabetes mellitus (NIDDM), and are equally neurotoxic . These, and other pathophysiological parallels, afford some insight into the probable cause of Alzheimer disease and, as such, forms the basis of the causal hypothesis advanced in this paper.

11/7/154 (Item 128 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10091391 BIOSIS NO.: 199598546309
Posterior hypothalamic deafferentation or 5,7-dihydroxytryptamine inhibit corticotropin-releasing hormone, ACTH and corticosterone responses following photic stimulation.
AUTHOR: Feldman Shaul(a); Weidenfeld Joseph

AUTHOR ADDRESS: (a)Dep. Neurol., Hadassah Univ. Hosp., Hebrew Univ.-Hadassah Med. Sch., POB 12000, Jerusalem 91120**Israel
JOURNAL: Neuroscience Letters 198 (2):p143-145 1995 ISSN: 0304-3940 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The effect of posterior hypothalamic deafferentation (PHD) or hypothalamic serotonin (5-HT) depletion by 5,7-dihydroxytryptamine on corticotropin-releasing hormone (CRH), ACTH and corticosterone (CS) responses, following photic stimulation, was investigated in male rats. In intact animals, photic stimulation caused median eminence (ME) CRH depletion and a rise in serum ACTH and CS levels. In rats with PHD, in which previous studies have demonstrated 5-HT depletion, or with neurotoxin induced hypothalamic 5-HT reduction, these responses were markedly inhibited. This indicates that the blockage in the release of ME CRH into the portal circulation in rats with hypothalamic 5-HT depletion is responsible for the inhibition of the pituitary -adrenocortical responses following photic stimulation.

11/7/158 (Item 132 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
10026655 BIOSIS NO.: 199598481573
Detection of MDMA neurotoxicity in vivo. PET studies in the living baboon brain.
AUTHOR: Scheffel U; Szabo Z; Mathews W B; Finley P A; Dannals R F; Ravert H T; Szabo K; Ricaurte G A
AUTHOR ADDRESS: Johns Hopkins Med. Inst., Baltimore, MD 21205**USA
JOURNAL: Society for Neuroscience Abstracts 21 (1-3):p861 1995 CONFERENCE/MEETING: 25th Annual Meeting of the Society for Neuroscience San Diego, California, USA November 11-16, 1995 ISSN: 0190-5295
RECORD TYPE: Citation LANGUAGE: English

11/7/179 (Item 153 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09764309 BIOSIS NO.: 199598219227
The anterior pituitary gland as a possible site of action of kainic acid.
AUTHOR: Zanisi Mariarosa(a); Galbiati Mariarita; Messi Elio; Martini Luciano
AUTHOR ADDRESS: (a)Inst. Endocrinology, via G. Balzaretti 9, 20133 Milano** Italy
JOURNAL: Proceedings of the Society for Experimental Biology and Medicine 206 (4):p430-437 1994
ISSN: 0037-9727 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The purpose of the present study was to analyze the direct effect of kainic acid (KA), an agonist of L-Glutamate, on the secretion of LH and FSH from anterior pituitary (AP) of male rats perfused in vitro. At low concentrations (1 mu-M), KA was able to stimulate the release of both gonadotropins from AP of 50-day-old male rats, but the response to subsequent stimuli was markedly impaired. This, however, was not due to a neurotoxic action of KA, but seemed rather suggestive of a down-regulation or desensitization of KA receptors. The stimulatory action of KA on LH and FSH secretion was age-dependent, since the agonist was completely ineffective on the AP of 75-day- and 18-month-old male rats. DNQX (6,7-dinitroquinoxaline-2,3-dione), a specific antagonist of the KA receptor subtype, was able to block the KA-induced gonadotropin secretion; similarly, AP-5 (2-amino-5-phosphonovalerate), a competitive NMDA receptor antagonist, prevented the stimulatory effect of KA on LH and FSH release. An interaction between the opiatergic and the excitatory aminoacid (EAA) systems emerged from the observation that pulses of KA applied to AP of 50-day-old male rats during a continuous perfusion with a medium containing morphine (5 mu-M) failed to increase gonadotropin secretion. These results indicate that KA can, at low concentrations, directly stimulate LH and FSH secretion by acting at AP level; this effect disappears with progression of age, and might be exerted both through NMDA and non-NMDA receptor subtypes. Finally, the results provide evidence that opioids and excitatory aminoacids might influence gonadotropin secretion from AP by acting in opposite directions.

11/7/192 (Item 166 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09661265 BIOSIS NO.: 199598116183
Effects of neurotoxic lesions in the posterior hypothalamic region on psychomotor activity and learning.
AUTHOR: Alvarez X A(a); Franco A; Fernandez-Novoa L; Cacabelos R
AUTHOR ADDRESS: (a)Dep. Biomed. Res., Inst. CNS Disorders, Basic Clin. Neurosci. Res. Cent., P.O. Box 733, E-15080-**Spain
JOURNAL: Agents and Actions 43 (1-2):p21-23 1994 ISSN: 0065-4299 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Histamine (HA) acts as a neurotransmitter and/or neuromodulator in mammalian brain. Central HA has been found to be involved in the regulation of behavioral, cognitive, neurovegetative, neuroendocrine and neuroimmune functions. In this study we have evaluated psychomotor activity (PMA) and passive avoidance behavior (PAB) in rats with bilateral neurotoxic lesions in the posterior hypothalamic region (PHR) (L), where histaminergic neurons are located, and in sham-operated rats (S), two weeks after neurosurgery. In an open-field paradigm, lesioned rats showed higher PMA scores than sham-operated animals. However, L rats exhibited a significant decrease in PMA on consecutive days (motor habituation) similar to that found in S rats. In a maze paradigm, in which the animals had to learn to stay on a neutral platform in order to avoid a 1.5 mA electric footshock during 10 trails, no significant differences were observed between L and S rats on the task performance. According to the present results, it seems that bilateral neurotoxic lesions in the PHR induced hyperactivity with no apparent effects on PAB, suggesting that neuronal HA might be involved, directly and/or by influencing arousal/alertness-mediated mechanisms, in the regulation of PMA processes.

11/7/195 (Item 169 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09636011 BIOSIS NO.: 199598090929
Effect of 6-hydroxydopamine injection into the arcuate hypothalamic nucleus on the osmotic release of vasopressin in conscious rats.
AUTHOR: Yamaguchi Ken'ichi(a); Hama Hitoshi; Watanabe Kazuo; Yamaya Kanemitsu
AUTHOR ADDRESS: (a)Dep. Physiol., Niigata Univ. Sch. Med., Asahimachi-Dori 1, Niigata City, Niigata 951**Japan
JOURNAL: European Journal of Endocrinology 131 (6):p658-663 1994 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The aim of the present study was to evaluate a role in vasopressin secretion of the catecholaminergic neurons, including the tuberohypophysial dopaminergic neurons situated in the arcuate hypothalamic nucleus. A neurotoxin , 6-hydroxydopamine (6 g/l), was injected locally into the arcuate nucleus and its effects on catecholamine levels of the hypothalamic tissue and the neurointermediate lobe, and on the plasma vasopressin concentrations before and during iv infusion (0.1 ml kg-1 min-1) of isotonic (0.15 mol/l) or hypertonic saline (2.5 mol/l), were examined in conscious rats. The infusion of hypertonic saline produced increases of plasma vasopressin 1 5 and 30 min later, accompanied by elevations of plasma osmolality, sodium, chloride and arterial pressure. The vasopressin response was potentiated markedly by the 6-hydroxydopamine injection performed 8 days before, which hardly affected the responses of the other variables. Histological examination indicated that the injection sites of 6-hydroxydopamine in those rats had been located in the area ranging from rostral to medial arcuate nucleus. The iv infusion of isotonic saline did not change plasma vasopressin, osmolality, sodium, chloride or arterial pressure, regardless of the presence or absence of pretreatment with 6-hydroxydopamine. it was confirmed that when 6-hydroxydopamine was injected into the arcuate nucleus region 8 days before, noradrenaline and adrenaline concentrations of the hypothalamic tissue containing the injection site were decreased remarkably, although we could not det any significant alteration in the dopamine concentration of the hypothalamic tissue or the neurointermediate lobe. On the bas these results, we concluded that catecholaminergic neurons in the arcuate nucleus may act to inhibit osmotic vasopressin secretion.

11/7/201 (Item 175 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09513979 BIOSIS NO.: 199497522349
Neurotoxin -stimulated hypothalamo - pituitary -adrenal (HPA) axis function in mice.
AUTHOR: Carino M(a); Giovanbaista B; Hilal R; Spinedi E
AUTHOR ADDRESS: (a)Neuroendocrine Unit, Dep. Neurosci., 1900 La Plata** Argentina
JOURNAL: Society for Neuroscience Abstracts 20 (1-2):p938 1994 CONFERENCE/MEETING: 24th Annual Meeting of the Society for Neuroscience Miami Beach, Florida, USA November 13-18, 1994 ISSN: 0190-5295 RECORD TYPE: Citation LANGUAGE: English

11/7/204 (Item 178 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09413204 BIOSIS NO.: 199497421574
Effects of neurotoxic lesions in histaminergic neurons on brain tumor necrosis factor levels.
AUTHOR: Alvarez X A(a); Franco A; Fernandez-Novoa L; Cacabelos R
AUTHOR ADDRESS: (a)Dep. Biomedical Res., Inst. CNS Disorders, Baisc Clinical Neurosci. Res. Center, PO Box 733, E-1**Spain
JOURNAL: Agents and Actions 41 (SPEC. CONF. ISSUE):pC70-C72 1994 ISSN: 0065-4299 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Histamine (HA) is a biogenic amine involved in the regulation of neurovegetative, cognitive, neuroendocrine a neuroimmune functions in the central nervous system (CNS). A bidirectional interaction between the CNS and the neuroim system has been demonstrated in recent years. However, data concerning brain HA-cytokine interactions are scarce. In this study we have evaluated tumor necrosis factor-alpha (TNF-alpha) levels in the posterior hypothalamic region (PHR) and hippocampus (HP) of rats with: (a) bilateral ibotenic acid neurotoxic lesions in histaminergic neurons located in the PHR (l); (b) saline injections in the PHR (S); and (c) sham operation (C), two weeks after neurosurgery. The bilateral disruption of PHR HA neurons with ibotenic acid decreased TNF-alpha levels in the PHR with respect to sham-operated (C), but not saline-injected (S), rats. In contrast, hippocampal TNF-alpha concentrations were higher in lesioned rats (l) than in C and S animals. Our results indicate that the neurotoxic destruction of HA neurons decreases TNF-alpha synthesis in the hypothalamus while enhancing TNF-alpha production in the hippocampus, suggesting that neuronal HA might be involved in the regulation of the brain TNF-alpha system.

11/7/206 (Item 180 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09354165 BIOSIS NO.: 199497362535
Evidence that noradrenergic neurons in the A1 and A2 nuclei are lesioned by low doses of 6-OHDA injected into the locus coeruleus.
AUTHOR: Engelbrecht A H(a); Russell V; Carstens M E; De Villiers A S; Searson A; Jaffer A; Taljaard J J F
AUTHOR ADDRESS: (a)MRC Res. Unit Neurochem. Mental Dis., Dep. Chem. Pathol., Univ. Stellenbosch, Tygerberg Hospital**South Africa

JOURNAL: Journal of Neuroscience Methods 52 (1):p57-60 1994 ISSN: 0165-0270 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: In order to determine the specificity of a lesion aimed at the locus coeruleus (LC), various doses of 6-hydroxydopamine (6-OHDA), a neurotoxin which selectively lesions catecholaminergic neurons, were bilaterally infused into the LC. The noradrenaline (NA) concentration in the frontal cortex; hippocampus. hypothalamus , LC, A1 and A2 nuclei decreased with increasing doses of 6-OHDA. A 1 mu-g dose of 6-OHDA injected bilaterally into the LC caused maximal depletion of the NA concentration in the frontal cortex, hippocampus and A1 and A2 nuclei. A dose of 2 mu-g 6-OHDA caused further depletion of the NA content of the hypothalamus and LC. These findings suggest that A1 and A2 neurons which project to the hypothalamus may have been lesioned or that the noradrenergic projection from the LC to the hypothalamus may be greater than was previously suspected. Alternatively, leakage of 6-OHDA into the cerebrospinal fluid may have occurred at the higher doses, thus directly exposing the hypothalamus to the toxic effects of 6-OHDA.

11/7/212 (Item 186 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09233877 BIOSIS NO.: 199497242247
Serotonergic neurotoxic lesions facilitate male sexual reflexes.
AUTHOR: Marson Lesley(a); McKenna Kevin E
AUTHOR ADDRESS: (a)Div. Urol., CB 7235, 427 Burnett-Womack Clin. Sci. Bldg., Univ. N.C. Sch. Med., Chapel Hill, NC **USA
JOURNAL: Pharmacology Biochemistry and Behavior 47 (4):p883-888 1994 ISSN: 0091-3057 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The effects of the neurotoxin 5,7 dihydroxytryptamine (5,7 DHT) on the urethrogenital reflex was examined in anesthetized male rats. Both ICV and intrathecal administration of 5,7 DHT produced a marked depletion (92%) of spinal 5-hydroxytryptamine (5-HT) and 5-hydroxyindole acetic acid (5-HT IAA) levels. ICV but not intrathecal administration of 5,7 DHT also caused a moderate reduction in 5-HT and 5-HT IAA levels in the medulla and hypothalamus (40-48%). No reduction in adrenergic levels were observed. In spinally intact, vehicle-treated rats the urethrogenital reflex could not be evoked. However, the urethrogenital reflex could be evoked in rats pretreated with either ICV or intrathecal 5,7 DHT prior to section of the spinal cord. These data support the hypothesis that 5-HT mediates the descending inhibition of male sexual reflexes.

11/7/228 (Item 202 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09000249 BIOSIS NO.: 199497008619
Ibotenic acid-induced lesions of the medial preoptic area/anterior hypothalamus enhance the display of progesterone-facilitated lordosis in male rats.
AUTHOR: Olster Deborah H
AUTHOR ADDRESS: Psychol. Dep., Univ. Calif., Santa Barbara, CA 93106**USA
JOURNAL: Brain Research 626 (1-2):p99-105 1993 ISSN: 0006-8993 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Electrical lesions of the medial preoptic area/anterior hypothalamus (MPOA/AH) have been reported to enhance the display of steroid-induced lordosis in castrated male rats. This study employed the cell body-specific neurotoxin , ibotenic acid, to ascertain whether neurons originating in this region (as opposed to axons of passage) tonically inhibit steroid-induced lordosis in adult male rats. Castrated, adult Long-Evans males received bilateral electrical lesions or injections of ibotenic acid or vehicle aimed at the MPOA/AH. Following administration of estradiol benzoate (EB) and progesterone, lordosis quotients (LQs) and lordosis ratings (LRs) were significantly higher in groups of rats with electrical lesions (LQ = 62.2 +- 15. 1; LR = 1.22 +- 0.34) and ibotenic acid-induced lesions (LQ = 58.1 +- 12.2; LR = 0.99 +- 0.24) than in the control group (LQ = 12.8 +- 7.3; LR 0.22 +- 0.13). To determine whether this enhancement of receptive behavior in MPOA/AH-lesioned males was an effect on estradiol-induced, as compared to progesterone-facilitated lordosis, groups of castrated rats in a second experiment received bilateral injections of ibotenic acid or vehicle aimed at the MPOA/AH and were tested for lordosis after administration of EB alone and again after injection of progesterone. Following treatment with EB alone, rats with ibotenic acid-induced MPOA/AH lesions tended to be slightly less receptive than control animals. However, following injections of progesterone, LQs and LRs were higher in the MPOA/AH-lesioned group than in the control animals, as had been observed in the first experiment. These data are consistent with the hypothesis that cell bodies, rather than axons of passage, originating in the MPOA/AH exert tonic inhibitory control over the display of progesterone-facilitated lordosis in adult male rats.

11/7/230 (Item 204 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
08999994 BIOSIS NO.: 199497008364
Serotonergic lesions decrease mu- and delta-opiate receptor binding in discrete areas of the hypothalamus and in the midbrain central gray.
AUTHOR: Allen Donald L(a); Johnson Allan E; Tempel Ann; Zukin R Suzanne; Luine Victoria N; McEwen Bruce S
AUTHOR ADDRESS: (a)Dep. Anat. Cell Biol., Univ. Cincinnati Med. Cent., 231 Bethesda Avenue, Cincinnati, OH 45267-05**USA
JOURNAL: Brain Research 625 (2):p269-275 1993 ISSN: 0006-8993 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Serotonergic nerve terminals in the brain were lesioned by intraventricular infusion of the selective neurotoxin 5,7-dihydroxytryptamine (5,7-DHT) and levels of mu- and delta-opiate binding were measured in brain areas implicated in reproductive behavior and gonadotropin secretion. The lesion decreased mu-receptor binding in the preoptic area (mPOA) and the midbrain central gray, while delta-receptor binding was decreased in the mPOA and the dorsomedial nucleus of the hypothalamus . Hypothalamic serotonergic lesions also attenuated morphine inhibition of female sexual behavior. These results indicate the existence of serotonergic-opiate interactions in select regions of the brain and suggest that these interactions may be important in the regulation of lordosis behavior.

11/7/231 (Item 205 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
08999935 BIOSIS NO.: 199497008305
A partial blockade of catecholaminergic neurotransmission with 6-hydroxydopamine decreases mRNA level of gonadotropin releasing hormone in the male rat hypothalamus .
AUTHOR: Kim Kyungjin(a); Lim In Sun; Cho Byung Nam; Kang Sang Soo; Lee Byung Ju; Choi Kyung Hee; Chung Chin Ha; Lee Chung Choo; Cho Wan Kyoo; Wuttke Wolfgang
AUTHOR ADDRESS: (a)Dep. Mol. Biol., Coll. Nat. Sci., Seoul Natl. Univ., Seoul 151-742**South Korea
JOURNAL: Neuroendocrinology 58 (1):p146-152 1993 ISSN: 0028-3835 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Central catecholamines (CA) are known to be involved in the regulation of synthesis and secretion of gonadotropin releasing hormone (GnRH) from the hypothalamus . However, no attempt has been yet made to determine whether CA affects GnRH expression. To this end, the effect of 6-hydrodopamine (6-OHDA), a catecholaminergic neurotoxin , on GnRH mRNA level was examined. Hypothalamic tissues obtained from adult male rats were incubated with medium containing 6-OHDA. To ensure the effect of 6-OHDA on CA depleting action, CA levels in media and in postincubation tissues were determined. Increasing concentrations of 6-OHDA resulted in decrease in norepinephrine (NE) and dopamine (DA) contents in a dose dependent manner. Tratment with 6-OHDA (5 times 10-4 M) produced a time-dependent decrease in NE but not DA, when CA levels in media were determined at 30 min intervals during the incubation period. To determine changes in GnRH mRNA level in response to 6-OHDA treatment in vitro, for 2.5 h total cytoplasmic RNA fractions were isolated from postincubation hypothalamic tissues and used for RNA-blot hybridization with 32P-labeled GnRH riboprobe. A blockade of CA neurotransmission with 6-OHDA (5 times 10-4 M) significantly reduced GnRH mRNA level by half over its control and internal control (actin mRNA) groups. Northern blot analysis revealed that addition of NE (1 times 10-6 M) reversed the decreased GnRH mRNA level by 6-OHDA. These studies clearly demonstrated that GnRH mRNA level was modified by the combination of 6-OHDA and/or NE in rat hypothalamic tissues incubated in vitro, indicating that CA neurotransmission influences GnRH gene expression.

11/7/232 (Item 206 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
08988933 BIOSIS NO.: 199396140434
Pressor response to microinjection of clonidine into the hypothalamic paraventricular nucleus in conscious rats.
AUTHOR: Ebihara Hiroaki; Kawasaki Hiromu; Nakamura Shigeru; Takasaki Koichiro(a); Wada Akihiko
AUTHOR ADDRESS: (a)Dep. Pharmacol., Miyazaki Med. Coll., 5200 Kiyotake, Miyazaki 889-16**Japan
JOURNAL: Brain Research 624 (1-2):p44-52 1993 ISSN: 0006-8993 DOCUMENT TYPE: Article
RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: We have reported that intracerebroventricular (i.c.v.) injection of clonidine causes pressor response in conscious rats. To determine the effective brain site, cardiovascular responses induced by unilateral microinjection of clonidine into various hypothalamic nuclei of conscious rats were studied. Microinjection of clonidine (5-20 mu-g/0.5 mu-l) into the paraventricular nucleus (PVN) of conscious rats dose-dependently produced a long-lasting pressor response with a decrease in heart rate, which mimicked the response to i.c.v. injection of clonidine. However, clonidine (10 mu-g) injection into various hypothalamic nuclei (anterior, posterior, ventromedial and dorsomedial nucleus) caused a small or no pressor response. In anesthetized rats, clonidine injected into the PVN induced a long-lasting depressor response concomitant with bradycardia. PVN pretreatment with the a-2-adrenoceptor antagonist, yohimbine (1 and 10 mu-g), dose-dependently inhibited the pressor response to PVN injection of clonidine, but the alpha-1-adrenoceptor antagonist, prazosin (1 mu-g), had no significant effect. Central (i.c.v.) pretreatment with the vasopressin (AVP) V-1-receptor antagonist, (d(CH-2)-5Tyr(Me))-AVP (0.5 and 2.0 mu-g), dose-dependently inhibited the pressor response to PVN injection of clonidine (10 mu-g), while systemic (i.v.) and local (intra-PVN injection) pretreatments with V-1-receptor antagonist (2.0 mu-g) had no effect. These results suggest that the pressor response to microinjection of clonidine into the PVN of conscious rats is mediated by endogenous brain AVP, which is released by activation of alpha-2-adrenoceptors. It is also suggested that the PVN is a possible brain site for the pressor response to i.c.v. injected clonidine.

11/7/251 (Item 225 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
08845829 BIOSIS NO.: 199395135180
Effects of intracerebroventricular and intrahypothalamic cocaine administration on adrenocortical secretion.
AUTHOR: Saphier David(a); Welch Jon E; Farrar Glenn E; Goeders Nick E
AUTHOR ADDRESS: (a)Dep. Pharmacology Therapeutics, Louisiana State University Medical Center, PO Box 33932, Shrevep**USA
JOURNAL: Neuroendocrinology 57 (1):p54-62 1993 ISSN: 0028-3835 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: Cocaine (COC) has been described as exerting potent stimulatory effects on the hypothalamo - pituitary - adrenocortical (HPA) axis. In the present study, we investigated the acute and chronic effects of intracerebroventricular and

intrahypothalamic injections of COC in rats. Twenty minutes following intracerebroventricular injection of COC (1-100 mu-g), dose-dependent increases in plasma corticosterone (CS) were observed, although the highest dose tested (100 mu-g) evoked a significantly smaller response than that following 50 mu-g. Prior stressing of the animals resulted in elevated plasma CS levels (315 +- 16 ng/ml) and significantly decreased plasma CS concentrations following 50 mu-g COC (87.8 +- 3.2%). Injections above the hypothalamic paraventricular nucleus (PVN), the site of corticotropin-releasing-factor-secreting neurons which regulate HPA activity, required relatively higher doses of COC in order to elicit increases in plasma CS; injections of 0.5 mu-g had no effect, 1 mu-g resulted in an increase to 168 +- 68 ng/ml (p lt 0.005), and 2.5 mu-g produced an increase to 146 +- 29 ng/ml (p lt 0.025). Post-PVN injections of COC, behind the posterior margin of the PVN in the vicinity of the ventral noradrenergic ascending bundle, also required a high dose (2.5 mu-g) in order to elicit a plasma CS response (208 +- 19 ng/ml; p lt 0.005), with no significant response seen following 0.5 mu-g COC. No effects of specific neurotoxic lesions of the catecholaminergic or serotonergic innervation of the hypothalamus were observed upon adrenocortical responses to COC. Chronic intracerebroventricular administration of COC (5 mu-g/day, 10 days) to conscious rats resulted in significant elevations in basal plasma CS (238 +- 36 ng/ml; p lt 0.025) above that found in saline-treated animals (129 +- 19 ng/ml). However, animals receiving daily COC injections were able to exhibit a similar CS response (336 +- 26 ng/ml) to that seen in the chronic saline-treated group given an acute intracerebroventricular COC challenge (328 +- 23 ng/ml). Neurochemical changes in monoamine metabolism were also measured in medial prefrontal cortex, hippocampus, and hypothalamus following single or repeated daily injections of COC, and these are discussed in the context of the data on adrenocortical responses. The results of this study demonstrate that while a local intrahypothalamic action of COC may elicit HPA activation, probably through a stimulation of catecholaminergic activity, the predominant site of action of COC in such activation appears to reside elsewhere in the central nervous system. The elevated plasma CS concentrations observed even in saline-challenged rats following chronic COC administration may reflect an anxiety state similar to that seen in chronic human COC users. At the same time, however, these changes do not appear to alter the responsiveness of the HPA axis to subsequent COC administration.

11/7/1 (Item 1 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
11892717 99335095 PMID: 10408610
Central serotonin depletion modulates the behavioural, endocrine and physiological responses to repeated social stress and subsequent c-fos expression in the brains of male rats.
Chung K K; Martinez M; Herbert J
Department of Anatomy, MRC Cambridge Centre for Brain Repair, University of Cambridge, UK.
Neuroscience (UNITED STATES) 1999, 92 (2) p613-25, ISSN 0306-4522 Journal Code: 7605074
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
Intraspecific confrontation has been used to study effect of depleting central serotonin on the adaptation of male rats to repeated social stress (social defeat). Four groups of adult male rats were used (serotonin depletion/sham: stressed; serotonin depletion/sham: non-stressed). Central serotonin was reduced (by 59-97%) by a single infusion of the neurotoxin 5,7-dihydroxtryptamine (150 microg) into the cerebral ventricles; levels of dopamine and noradrenaline were unaltered (rats received appropriate uptake blockers prior to neurotoxic infusions). Sham-operated animals received solute only. Rats were then either exposed daily for 10 days to a second larger aggressive male in the latter's home cage, or simply transferred to an empty cage (control procedure). Rats with reduced serotonin failed to show the increased freezing behaviour during the pre-defeat phase of the social interaction test characteristic of sham animals. There was no change in the residents' behaviour. Core temperature increased during aggressive interaction in sham rats, and this did not adapt with repeated stress. By contrast, stress-induced hyperthermia was accentuated in serotonin-reduced rats as the number of defeat sessions increased. Basal core temperature was unaffected by serotonin depletion. Heart rate increased during social defeat, but this did not adapt with repeated stress; serotonin depletion had no effect on this cardiovascular response. Basal corticosterone was increased in serotonin-depleted rats, but the progressive reduction in stress response over days was not altered. C-fos expression in the brain was not altered in control (non-stressed) rats by serotonin reduction in the areas examined, but there was increased expression after repeated social stress in the medial amygdala of 5-HT depleted rats. These experiments show that reduction of serotonin alters responses to repeated social stress in male rats, and suggests a role for serotonin in the adaptive process. Record Date Created: 19990917 Record Date Completed: 19990917

11/7/260 (Item 234 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
08775559 BIOSIS NO.: 199395064910
Effects of intracisternal vs. intrahypothalamic 5,7-DHT on feeding elicited by hypothalamic infusion of NE.
AUTHOR: Coscina Donald V(a); De Rooy Elizabeth C H
AUTHOR ADDRESS: (a)Sect. Biopsychol., Clarke Inst. Psychiatry, 250 College St., Toronto, Ont. M5T 1R8**Canada
JOURNAL: Brain Research 597 (2):p310-320 1992 ISSN: 0006-8993 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: A variety of evidence has led to suggestions that brain serotonin (5-HT) and norepinephrine (NE) interact within the medial hypothalamus to control food intake. To test the possibility that chronic decrements in 5-HT might enhance NE-induced feeding, adult male rats were prepared with permanently indwelling cannulae aimed at the paraventricular nucleus (PVN), then received either intracisternal (IC) or PVN injections of the 5-HT neurotoxin , 5,7-dihydroxytryptamine (5,7-DHT) vs. its vehicle, 1% ascorbic acid. Over a 4-week period, IC-5,7-DHT rats showed no signs of enhanced daily feeding or drinking. However, in 40-min intake tests, feeding but not drinking was enhanced by injecting 20 nmol NE into the PVN commencing 2 weeks after neurotoxin treatment. Terminal monoamine assays confirmed that IC-5,7-DHT produced large (80-90%) depletions of brain regional 5-HT. A functional index of 5-HT terminal damage was also implied by the impaired short-term feeding responses IC-5,7-DHT rats showed to the systemic administration of the 5-HT-IA agonist, 8-hydroxy-2-(di-n-propylamino) tetralin (8-OH-DPAT) when tested between 3 and 4 weeks after IC treatment. Over a comparable 4-week period, PVN-5,7-DHT rats also showed no tendencies to overeat or overdrink on a daily basis. However, in contrast to IC-5,7-DHT rats, they also showed no differences in their feeding or drinking responses to NE injections into the PVN. This was so despite reliable depletions of 5-HT in the hypothalamus (-28%) and hippocampus (-71%). These results support earlier work showing that neither widespread nor localized hypothalamic damage to brain 5-HT neurons produce chronic overeating. However, the data suggest that phasic enhancements of PVN NE activity may trigger enhanced feeding when there is widespread damage to brain 5-HT neurons, although the PVN does not appear to be the brain site mediating this effect.

11/7/265 (Item 239 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
08511011 BIOSIS NO.: 199344061011
5,7-DHT infusions into the medial hypothalamus facilitate lordosis in preweanling rats.
AUTHOR: Benedict G S(a); Williams C L
AUTHOR ADDRESS: (a)Dep. Psychol., Columbia Univ., New York, N.Y. 10027**USA
JOURNAL: Society for Neuroscience Abstracts 18 (1-2):p888 1992 CONFERENCE/MEETING: 22nd Annual Meeting of the Society for Neuroscience Anaheim, California, USA October 25-30, 1992 ISSN: 0190-5295 RECORD TYPE: Citation LANGUAGE: English

11/7/267 (Item 241 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
07415865 BIOSIS NO.: 000040030174
IN-VITRO MODULATION OF BETA ENDORPHIN SECRETION FROM RAT NEUROINTERMEDIATE PITUITARY AFTER HYDROXYDOPAMINE-INDUCED DEGENERATION OF NERVE TERMINALS
AUTHOR: SALAND L C; CARR J A; SAMORA A; BENAVIDEZ S
AUTHOR ADDRESS: DEP. ANATOMY, UNIV. N.M. SCH. MED., ALBUQUERQUE, N.M. 87131.
JOURNAL: THIRTIETH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CELL BIOLOGY, SAN DIEGO, CALIFORNIA, USA, DECEMBER 9-13, 1990. J CELL BIOL 111 (5 PART2). 1990. 340A. 1990
CODEN: JCLBA DOCUMENT TYPE: Meeting RECORD TYPE: Citation LANGUAGE: ENGLISH

11/7/270 (Item 244 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
05622786 BIOSIS NO.: 000083095927
NEUROENDOCRINE AND BEHAVIORAL EFFECTS OF INTRATHECAL CAPSAICIN IN ADULT FEMALE RATS
AUTHOR: NANCE D M; KING T R; NANCE P W
AUTHOR ADDRESS: DEP. ANATOMY, DALHOUSIE UNIV., HALIFAX, NOVA SCOTIA B3H 4H7, CANADA.
JOURNAL: BRAIN RES BULL 18 (1). 1987. 109-114. 1987 FULL JOURNAL NAME: Brain Research Bulletin CODEN: BRBUD RECORD TYPE: Abstract LANGUAGE: ENGLISH
ABSTRACT: Neural feedback from the gonads and the reproductive tract has an integral role in normal reproductive function in female rats. To further assess the role of sensory feedback in neuroendocrine control, the effects of an intrathecal injection of the neurotoxin , capsaicin (100-125 .mu.g) into the lumbosacral region of the spinal cord on reproductive function was tested in female rats. In addition, the effect of capsaicin on the response to noxious heat and pressure were tested. Intrathecal capsaicin had no effect on estrous cycles, ovarian compensatory hypertrophy or female sexual behavior. However, capsaicin treated animals showed a dramatic reduction in fertility, relative to vehicle treated control rats and this was shown to be due to the reduced capacity of vaginal/cervical stimulation to produce pseudopregnancy. Consistent with a selective loss in the effectiveness of cervical stimulation, immobilization produced by vaginal probing was reduced in capsaicin treated rats, but response slowly recovered across time in some animals. Similarly, the ability of vaginal probing to induce a lordotic response during estrogen treatment was reduced in the capsaicin treated animals. The capsaicin treated animals showed analgesia to noxious heat, as measured by the tail flick test, but showed a normal foot withdrawal in response to pressure. The analgesic effect of vaginal stimulation on noxious pressure was unaltered in the capsaicin treated rats. Finally, the neurotoxin effect of capsaicin was verified in terms of a quantitative reduction in substance P immunoreactivity in the dorsal horns of the lumbosacral region of the spinal cord of capsaicin treated rats, relative to control animals. Estrogen treatment of ovariectomized rats quantitatively reduced substance P immunoreactivity in the spinal cord, an effect even more apparent in the capsaicin treated animals. These studies indicate that intrathecal capsaicin has an effect in adult female rats that is specific to the endocrine and behavioral consequences of vaginal stimulation. Whether substance P mediates any of the endocrine or behavioral effects of cervical stimulation remains to be established.

11/7/274 (Item 248 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
04599031 BIOSIS NO.: 000079012068
EFFECT OF D L-ALPHA AMINOADIPATE ON THE MEDIOBASAL HYPOTHALAMUS AND ENDOCRINE FUNCTION IN THE RAT
AUTHOR: BRUNI J E; VRIEND J

AUTHOR ADDRESS: DEPARTMENT OF ANATOMY, UNIVERSITY OF MANITOBA, 730 WIL LIAM AVENUE, WINNIPEG, MANITOBA, CANADA R3E 0W3.
JOURNAL: ACTA NEUROPATHOL 64 (2). 1984. 129-138. 1984 FULL JOURNAL NAME: Acta Neuropathologica CODEN: ANPTA RECORD TYPE: Abstract LANGUAGE: ENGLISH
ABSTRACT: Using the glutamate analog, D,L-.alpha.-aminoadipic acid (D,L-.alpha.AA), experiments were conducted to examine the nature, extent and specificity of its toxicity in the mediobasal hypothalamus and to determine its effect on endocrine homeostasis. Neonatal rats received daily injections of D,L-.alpha.AA (4 g/kg BW [body wt]) on postnatal days 5-10 and were killed at various post-treatment intervals. Sex-matched littermates were given equimolar amounts of NaCl and served as controls. Treated rats killed 18 days post injection weighed slightly less than controls and had reduced testicular, ovarian and uterine weights, but the differences were not statistically significant. In D,L-.alpha.Aa treated rats serum and pituitary levels of TSH and PRL [prolactin] were comparable to control values. Pituitary content of LH [lutenizing hormone] (.male.s and .tau.'s)and FSH (.tau.'s), however, was lower ($P < 0.05$) in D,L-.alpha.AA treated rats than in controls, but serum levels were not significantly different. Distinct cytopathologic changes were evident in the arcuate nucleus and median eminence of D,L-.alpha.Aa-treated rats killed at 2 and 6 h post injection only. By 12 h evidence of acute damage had largely disappeared. Both glial and ependymal cells underwent edematous swelling and necrosis, but neurons were largely unaffected. Evidence of reactive changes, such as gliosis, infiltration of microglia and removal of debris, however, were not very conspicious. A random sample of mediobasal hypothalami of rats killed 18 days post injection failed to show any detectable lesion or residual effects of earlier pathology. Age at the time of exposure to the gliotoxin was found to be an important variable affecting both extent and duration of injury. The most deleterious effects were observed when the gliotoxin was administered in the form of a single injection on postnatal day 5 only. Normal neuronal activity and endocrine homeostasis, specifically gonadotropin, may be irreversibly altered as a consequence of transient disruption of the glial compartment.

11/7/283 (Item 257 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
02646570 BIOSIS NO.: 000067034632
ACUTE ELEVATIONS OF SERUM LUTEINIZING HORMONE INDUCED BY KAINIC-ACID N METHYL ASPARTIC-ACID OR HOMO CYSTEIC-ACID
AUTHOR: PRICE M T; OLNEY J W; CICERO T J
AUTHOR ADDRESS: DEP. PSYCHIATRY, WASH. UNIV. SCH. MED., 4940 AUDOBON AVE., ST. LOUIS, MO. 63110, USA.
JOURNAL: NEUROENDOCRINOLOGY 26 (6). 1978 352-358. 1978 FULL JOURNAL NAME: Neuroendocrinology CODEN: NUNDA RECORD TYPE: Abstract LANGUAGE: ENGLISH
ABSTRACT: It has been previously demonstrated that glutamate (GLU), a neuroexcitatory amino acid which destroys neurons in the arcuate nucleus of the hypothalamus (AH) when administered in high s.c. doses to rodents, induces an acute elevation of serum luteinizing hormone (LH) when adminstered in non-toxic doses, i.e., doses lower than those required to damage AH neurons. A neuroexcitatory mechanism (excitation of AH neurons) possibly underlies the acute rise in serum LH induced by GLU. To explore this hypothesis, several potent excitatory analogs of GLU were administered: kainic acid (KA), N-methyl-aspartic acid (NMA) and homocysteic acid (HCA) - in low doses to 25 day old male rats and found that each analog mimics GLU in producing acute elevations of serum LH. The lowest doses effective (LED) in inducing significant elevations in LH were: 0.03 .mu.M[moles]/g KA, 0.1 .mu.M/g NMA and 0.3 .mu.M/g HCA. Thus, the order of potencies (KA > NMA > HCA) for inducing LH elevations was the same as has been shown for either the neuroexcitatory or neurotoxic activities of these compounds. With the possible exception of KA, each compound induced LH elevations at 1/3-1/4 the lowest effective toxic doses (LETD). These potent neuroexcitatory analogs of GLU may readily penetrate AH from blood and stimulate firing of AH neurons which triggers discharge of luteinizing hormone-releasing hormone (LH-RH) into portal blood to give rise to pituitary output of LH. These compounds may be useful tools for studying mechanisms of neuroendocrine regulation.

11/7/284 (Item 258 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
02385891 BIOSIS NO.: 000065042927
ANALYSIS OF THE DISRUPTION IN HYPOTHALAMIC PITUITARY REGULATION IN RATS TREATED NEO NATALLY WITH MONO SODIUM L GLUTAMATE EVIDENCE FOR THE INVOLVEMENT OF TUBERO INFUNDIBULAR CHOLINERGIC AND DOPAMINERGIC SYSTEMS IN NEURO ENDOCRINE REGULATION
AUTHOR: NEMEROFF C B; KONKOL R J; BISSETTE G; YOUNGBLOOD W; MARTIN J B; BRAZEAU P; RONE M S; PRANGE A J JR; BREESE G R; ET AL.
AUTHOR ADDRESS: DIV. HEALTH AFFAIRS, BIOL. SCI. RES. CENT., UNIV. N.C., CHAPEL HILL, N.C. 27514, USA.
JOURNAL: ENDOCRINOLOGY 101 (2). 1977 613-622. 1977 FULL JOURNAL NAME: Endocrinology CODEN: ENDOA RECORD TYPE: Abstract LANGUAGE: ENGLISH
ABSTRACT: Adult rats which received monosodium-L-glutamate (MSG) (4 mg/g body wt) on alternate days for the first 10 days of life acquired neurotoxic lesions of the retina and arcuate nucleus and manifested an endocrine deficiency syndrome characterized by stunted growth, obesity, hypothyroidism, hypogonadism and pituitary atrophy. The biochemical basis for the MSG-induced endocrine dysfunction was examined and the findings of note were as follows: normal serum levels of TSH [thyrotropin stimulating hormone] and LH [luteinizing hormone] despite hypothyroidism and gonadal atrophy, and significantly reduced serum GH [growth hormone] levels in both males and females; elevated serum PRL [prolactin] levels in males, but females, normal or augmented pituitary release of LH and TSH to exogenous LHRH [LH releasing hormone] and TRH [thyrotropin releasing hormone]. Within the central nervous system: a normal diurnal rhythm of pineal N-acetyltransferase activity despite optic atrophy; normal concentrations of LHRH, TRH and somatostatin within the medial basal hypothalamus ; normal concentrations of norepinephrine (NE), choline acetyltransferase (CAT) and dopamine (DA) in all extrahypothalamic regions examined; normal concentrations of serotonin (5HT) and NE, but greatly reduced concentrations of DA (40-50%) and CAT activity (70-75%) in the arcuate nucleus (ARC) and median eminence (ME) of the hypothalamus . From these findings several conclusions were drawn: The MSG-induced endocrine deficiency syndrome appears to result from the destruction of ARC-ME dopaminergic and cholinergic tuberoinfundibular systems within the hypothalamus ; a normal concentration of serotonergic and noradrenergic neurons within the hypothalamus does not insure normal central neuroendocrine regulation; no more than 50% of the dopaminergic terminals in the ME arise from ARC perikarya; cell bodies within the ARC contribute very few, if any, nerve terminals containing releasing factors to the ME; MSG destroys the primary optic tracts while sparing the retino- hypothalamic projection; LHRH, somatostatin and TRH are not contained within cholinergic nerve terminals in the ME.

15/6/1 (Item 1 from file: 155) 09679475 21471103 PMID: 11587471
Abrupt-onset oculomotor paralysis: an endocrine emergency. Sep 2001

15/6/2 (Item 2 from file: 5) 09024104 BIOSIS NO.: 199497032474
Botulinum neurotoxin C1 blocks neurotransmitter release by means of cleaving HPC-1/syntaxin. 1993

15/6/3 (Item 3 from file: 5) 13582108 BIOSIS NO.: 200200210929
Botulinum toxin injection and surgical crico-myotomy for oro-pharyngeal dysphagia: Preliminary results of a phase-II study. 2001

15/6/4 (Item 4 from file: 5) 08975147 BIOSIS NO.: 199396126648
The cerebrospinal fluid in patients with moyamoya disease (spontaneous occlusion of the circle of Willis) contains high level of basic fibroblast growth factor. 1993

15/6/5 (Item 5 from file: 155) 11901760 99344722 PMID: 10416233
[Sotos syndrome associated with focal dystonia] Sindrome de Sotos asociado a distonia focal. May 16-31 1999

15/6/6 (Item 6 from file: 5) 08716384 BIOSIS NO.: 199395005735
Tetanus toxin and botulinum toxins type A and B inhibit glutamate, gamma-aminobutyric acid, aspartate, and methionine-enkephalin release from synaptosomes: Clues to the locus of action. 1992

15/6/7 (Item 7 from file: 5) 09720145 BIOSIS NO.: 199598175063
Vesicle-associated membrane protein-2 (synaptobrevin-2) forms a complex with synaptophysin. 1995

15/6/8 (Item 8 from file: 5) 10701696 BIOSIS NO.: 199799322841
Two component of glutamate exocytosis differentially affected by presynaptic modulation. 1996

15/6/9 (Item 9 from file: 5) 13693724 BIOSIS NO.: 200200322545
2002 Annual Meeting of the British Paediatric Neurology Association, Newcastle upon Tyn 2001

15/7/2 (Item 2 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
09024104 BIOSIS NO.: 199497032474
Botulinum neurotoxin C1 blocks neurotransmitter release by means of cleaving HPC-1/syntaxin.
AUTHOR: Blasi Juan; Chapman Edwin R; Yamasaki Shiji; Binz Thomas; Niemann Heiner; Jahn Reinhard(a)
AUTHOR ADDRESS: (a)Howard Hughes Med. Inst., Boyer Cent. Mol. Med., Yale Univ. Med. Sch., POB 9812, New Haven, CT 0**USA
JOURNAL: EMBO (European Molecular Biology Organization) Journal 12 (12):p 4821-4828 1993
ISSN: 0261-4189 DOCUMENT TYPE: Article RECORD TYPE: Abstract LANGUAGE: English
ABSTRACT: The anaerobic bacterium Clostridium botulinum produces several related neurotoxins that block exocytosis of synaptic vesicles in nerve terminals and that are responsible for the clinical manifestations of botulism . Recently, it was reported that botulinum neurotoxin type B as well as tetanus toxin act as zinc-dependent proteases that specifically cleave synaptobrevin, a membrane protein of synaptic vesicles (Link et al., Biochem. Biophys. Res. Commun., 189, 1017-1023; Schiavo et al., Nature, 359, 832-835). Here we report that inhibition of neurotransmitter release by botulinum neurotoxin type C1 was associated with the proteolysis of HPC-1 (= syntaxin), a membrane protein present in axonal and synaptic membranes. Breakdown of HBC-1/syntaxin was selective since no other protein degradation was detectable. In vitro studies showed that the breakdown was due to a direct interaction between HPC-1/syntaxin and the toxin light chain which acts as a metalloendoprotease. Toxin-induced cleavage resulted in the generation of a soluble fragment of HPC-1/syntaxin that is 2-4 kDa smaller than the native protein. When HPC1/syntaxin was translated in vitro, cleavage occurred only when translation was performed in the presence of microsomes, although a full-length product was obtained in the absence of membranes. However, susceptibility to toxin cleavage was restored when the product of membrane-free translation was subsequently incorporated into artificial proteoliposomes. In addition, a translated form of HPC-1/syntaxin, which lacked the putative transmembrane domain at the C-terminus, was soluble and resistant to toxin action. We conclude that HPC-1/ syntaxin is involved in exocytotic membrane fusion. Furthermore, these results indicate that the botulinum neurotoxin have evolved as related proteins recognizing the exocytotic fusion machine whereby individual toxin components target different components of the membrane fusion complex.

18/6/1 (Item 1 from file: 155) 09679475 21471103 PMID: 11587471
Abrupt-onset oculomotor paralysis: an endocrine emergency. Sep 2001

18/6/2 (Item 2 from file: 155) 14377514 22309002 PMID: 12422076
Blepharospasm in bardet-biedl syndrome: a case report. 2002

18/6/3 (Item 3 from file: 5) 11130480 BIOSIS NO.: 199799751625
Botulinum neurotoxin F, a VAMP-specific endopeptidase, inhibits Ca-2+-stimulated GH secretion from rat pituitary cells. 1997

18/6/4 (Item 4 from file: 155) 11089137 97444412 PMID: 9299640
Botulinum neurotoxin F, a VAMP-specific endopeptidase, inhibits Ca(2+)-stimulated GH secretion from rat pituitary cells. Jul 23 1997

18/6/5 (Item 5 from file: 5) 08940358 BIOSIS NO.: 199396091859
Botulinum toxin: Preferred treatment for hemifacial spasm. 1993

18/6/6 (Item 6 from file: 155) 10795121 97084604 PMID: 8930938
Characterization and distribution of SNARE proteins at neuroendocrine nerve endings. Nov 1996

18/6/7 (Item 7 from file: 155) 10735121 97084604 PMID: 8930938
Characterization and distribution of SNARE proteins at neuroendocrine nerve endings. Nov 1996

18/6/8 (Item 8 from file: 5) 01919946 BIOSIS NO.: 000062010040
CONTRACTION AND RELAXATION OF THE RETRACTOR PENIS MUSCLE AND PENILE ARTERY OF THE BULL A STUDY OF EFFECTS OF DRUGS AND TRANS MURAL NERVE STIMULATION ON ISOLATED SMOOTH MUSCLE STRIPS 1974

18/6/9 (Item 9 from file: 155) 03965493 83094030 PMID: 7348919
[Central nervous system diseases in cattle. 2. Diseases in young and adult cattle] Erkrankungen des Zentralnervensystems beim Rind. 2. Die Krankheiten jugendlicher und erwachsener Rinder. 1981

18/6/10 (Item 10 from file: 5) 11011461 BIOSIS NO.: 199799632606
Electroporation of botulinum neurotoxin serotypes A and B into GH-3 cells: Substrate cleavage and growth hormone secretion. 1996

18/6/11 (Item 11 from file: 5) 03426114 BIOSIS NO.: 000022069210
EPIDEMIOLOGIC REVIEWS VOL. 3 1981

18/6/12 (Item 12 from file: 155) 02537943 77229933 PMID: 18567
Evidence that acetylcholine releases noradrenaline in the sympathetic fibre. Jun 1977

18/6/13 (Item 13 from file: 155) 08137503 94203372 PMID: 8152548
Exploring the functional domain and the target of the tetanus toxin light chain in neurohypophysial terminals. Jan 1994

18/6/14 (Item 14 from file: 155) 10489312 96298912 PMID: 8741217
Expression of synaptosomal-associated protein SNAP-25 in endocrine anterior pituitary cells. Apr 1996

18/6/15 (Item 15 from file: 5) 01560012 BIOSIS NO.: 000011060001
HORMONES AND OSMO REGULATION IN MARINE BIRDS 1975

18/6/16 (Item 16 from file: 5) 14002542 BIOSIS NO.: 200200631363
Intractable cluster headaches in a migraineur. 2002

18/6/17 (Item 17 from file: 155) 10929051 97281275 PMID: 9135583
Involvement of SNAP-25 in TRH-induced exocytosis in pituitary GH4C1 cells. Apr 1997

18/6/18 (Item 18 from file: 5) 10895355 BIOSIS NO.: 199799516500
Involvement of SNAP-25 in TRH-induced exocytosis in pituitary GH4C1 cells. 1997

18/6/19 (Item 19 from file: 5) 13039489 BIOSIS NO.: 200100246638
Potassium channel inhibition by G-protein coupled receptors through Rho-dependent signaling. 2001

18/6/20 (Item 20 from file: 155) 05507779 87186703 PMID: 3494493
Localized diseases of the bovine brain and spinal cord. Mar 1987

18/6/21 (Item 21 from file: 5) 13082159 BIOSIS NO.: 200100289308
The neuropeptide PACAP38 exerts its antiproliferative effect by inhibiting the small GTpase RhoA. 2001

18/6/22 (Item 22 from file: 155) 05960468 88315035 PMID: 3137228
Purification and properties of the cytosolic substrate for botulinum ADP-ribosyltransferase. Identification as an Mr 22,000 guanine nucleotide-binding protein. Sep 5 1988

18/6/23 (Item 23 from file: 5) 06272656 BIOSIS NO.: 000086106839
PURIFICATION AND PROPERTIES OF THE CYTOSOLIC SUBSTRATE FOR BOTULINUM ADP-RIBOSYLTRANSFERASE IDENTIFICATION AS AN M-R 22000 GUANINE NUCLEOTIDE-BINDING PROTEIN 1988

18/6/24 (Item 24 from file: 155) 09839243 21650335 PMID: 11790300
Rac and Rho mediate opposing hormonal regulation of the ether-a-go-go-related potassium channel. Jan 8 2002

18/6/25 (Item 25 from file: 5) 13538319 BIOSIS NO.: 200200167140
Rac and Rho mediate opposing hormonal regulation of the ether-a-go-go-related potassium channel. 2002

18/6/26 (Item 26 from file: 155) 10586395 96401263 PMID: 8807639
Rho proteins are localized with different membrane compartments involved in vesicular trafficking in anterior pituitary cells. May 31 1996

18/6/27 (Item 27 from file: 5) 10464354 BIOSIS NO.: 199699085499
Rho proteins are localized with different membrane compartments involved in vesicular trafficking in anterior pituitary cells. 1996

18/6/28 (Item 28 from file: 155) 07029648 91270503 PMID: 2097523
Release of vasopressin from isolated permeabilized neurosecretory nerve terminals is blocked by the light chain of botulinum A toxin. 1990

18/6/29 (Item 29 from file: 155) 08975735 20266416 PMID: 10792045
Rapid regulated dense-core vesicle exocytosis requires the CAPS protein. May 9 2000

18/6/30 (Item 30 from file: 5) 12512229 BIOSIS NO.: 200000265731
Rapid regulated dense-core vesicle exocytosis requires the CAPS protein. 2000

18/6/31 (Item 31 from file: 5) 13693724 BIOSIS NO.: 200200322545
2002 Annual Meeting of the British Paediatric Neurology Association, Newcastle upon Tyne, UK. 2001

18/7/4 (Item 4 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
11089137 97444412 PMID: 9299640
Botulinum neurotoxin F, a VAMP-specific endopeptidase, inhibits Ca(2+)-stimulated GH secretion from rat pituitary cells.
Jacobsson G; Hakansson M L; Hulting A L; Meister B
Department of Neuroscience, karolinska Institute, Stockholm, Sweden.
Regulatory peptides (NETHERLANDS) Jul 23 1997, 71 (1) p37-44, ISSN 0167-0115 Journal Code: 8100479
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
Botulinum neurotoxin F (BoNTx F) is a zinc-dependent endopeptidase that causes proteolytic cleavage of the vesicle protein VAMP (vesicle-associated membrane protein). VAMP is an important component of the molecular machinery regulating docking and fusion of secretory vesicles with the target membrane. We have investigated presence of VAMP protein in cultured rat anterior pituitary cells. Confocal laser microscopy revealed presence of VAMP-like immunoreactivity in secretory granules of GH-containing cultured rat anterior pituitary cells. Using BoNTx F, we have investigated whether VAMP is involved in growth hormone (GH) secretion. Treatment of streptolysin-O permeabilized GH-secreting cells with BoNTx F (2.0 and 20 nM) significantly inhibited Ca(2+)-induced GH release. The results show that the secretory granules of rat anterior pituitary cell contain VAMP protein and suggest that VAMP is of importance in regulating Ca(2+)-mediated GH secretion. Record Date Created: 19971223 Record Date Completed: 19971223

18/7/6 (Item 6 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
10795121 97084604 PMID: 8930938
Characterization and distribution of SNARE proteins at neuroendocrine nerve endings.
Jurgutis P; Shuang R; Fletcher A; Stuenkel E L
Department of Physiology, University of Michigan, Ann Arbor 48109-0622, USA.
Neuroendocrinology (SWITZERLAND) Nov 1996, 64 (5) p379-92, ISSN 0028-3835 Journal Code: 0035665
Contract/Grant No.: NS31888; NS; NINDS Document type: Journal Article Languages: ENGLISH
Main Citation Owner: NLM Record type: Completed
Substantial evidence now exists to support a defined complex of interacting proteins, comprised of soluble, vesicle and plasma membrane components, as the core of a general membrane fusion mechanism. Specializations to the general secretory model occur based on cell-specific differences in Ca2+ regulation, secretory organelle types and secretory dynamics. The variation in secretory properties may also result, in part, from isoform diversity and selective-pairing of the molecular components of the core complex. The present report attempts to identify the SNARE proteins found in isolated peptidergic nerve endings of the rat neurohypophysis. The results demonstrate the presence of synaptosomal-associated protein of 25 kD, syntaxin and synaptobrevin as membrane-associated proteins in these nerve endings. Furthermore, we have utilized sucrose density gradient subcellular fractionation and immunoprecipitation protocols to investigate the synaptobrevin isotypes present on secretory granules and to probe using electrophysiological methods their functional relationship to secretion. Secretory granules were found to contain only the synaptobrevin 2 isoform, although the nerve endings themselves were found to possess in addition, synaptobrevin 1 and the closely related protein cellubrevin. Analysis of the secretory characteristics of single nerve endings using membrane capacitance measurements together with Botulinum B toxin dialysis demonstrated the critical importance of synaptobrevin 2 to both the rapid exocytotic release and a slower secretory process, that perhaps includes secretory granule recruitment and priming, in these peptidergic nerve endings. Record Date Created: 19970220 Record Date Completed: 19970220

18/7/10 (Item 10 from file: 5) IALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
11011461 BIOSIS NO.: 199799632606
Electroporation of botulinum neurotoxin serotypes A and B into GH-3 cells: Substrate cleavage and growth hormone secretion
AUTHOR: Foster K A(a); Duggan M J; Boyd R S
AUTHOR ADDRESS: (a)Speywood Lab. Ltd., St. George's Hosp. Med. Sch., Cranmer Terrace, Tooting, London SW17 0QS**UK
JOURNAL: Zentralblatt fuer Bakteriologie Supplement 28 (0):p216-217 1996 CONFERENCE/MEETING: Seventh European Workshop on Bacterial Protein Toxins Hindsgavl, Denmark July 2-7, 1995 ISSN: 0941-018X RECORD TYPE: Citation
LANGUAGE: English

18/7/11 (Item 11 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
03426114 BIOSIS NO.: 000022069210
EPIDEMIOLOGIC REVIEWS VOL. 3
AUTHOR: NATHANSON N; GORDIS L
AUTHOR ADDRESS: DEP. MICROBIOL., SCH. MED., UNIV. PA., PHILADELPHIA, PA. 19104, USA.
JOURNAL: NATHANSON, N. AND L. GORDIS (ED.). EPIDEMIOLOGIC REVIEWS, VOL. 3. VI+253P. JOHNS HOPKINS UNIVERSITY PRESS: BALTIMORE, MD., USA; LONDON, ENGLAND. ILLUS. MAPS. PAPER. ISBN 0-8018-2735-3(PAPER) ISBN 0-8018-2734-5(CLOTH). 0 (0). 1981. VI+253P. 1981 CODEN: EPIRD DOCUMENT TYPE: Book RECORD TYPE: Citation
LANGUAGE: ENGLISH

18/7/13 (Item 13 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
08137503 94203372 PMID: 8152548
Exploring the functional domain and the target of the tetanus toxin light chain in neurohypophysial terminals.
Dayanithi G; Stecher B; Hohne-Zell B; Yamasaki S; Binz T; Weller U; Niemann H; Gratzl M
Laboratoire de Neurobiologie Endocrinologique URA 1197, CNRS, Universite Montpellier-2, France.
Neuroscience (ENGLAND) Jan 1994, 58 (2) p423-31, ISSN 0306-4522 Journal Code: 7605074
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
The tetanus toxin light chain blocks calcium induced vasopressin release from neurohypophysial nerve terminals. Here we show that histidine residue 233 within the putative zinc binding motif of the tetanus toxin light chain is essential for the inhibition of exocytosis, in the rat. The zinc chelating agent dipicolinic acid as well as captopril, an inhibitor of zinc-dependent peptidases, counteract the effect of the neurotoxin. Synthetic peptides, the sequences of which correspond to motifs present in the cytoplasmic domain of the synaptic vesicle membrane protein synaptobrevin 1 and 2, prevent the effect of the tetanus toxin light chain. Our results indicate that zinc bound to the zinc binding motif constitutes the active site of the tetanus toxin light chain. Moreover they suggest that cleavage of synaptobrevin by the neurotoxin causes the inhibition of exocytotic release of vasopressin from secretory granules. Record Date Created: 19940511 Record Date Completed: 19940511

18/7/22 (Item 22 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
05960468 88315035 PMID: 3137228
Purification and properties of the cytosolic substrate for botulinum ADP-ribosyltransferase. Identification as an Mr 22,000 guanine nucleotide-binding protein.
Morii N; Sekine A; Ohashi Y; Nakao K; Imura H; Fujiwara M; Narumiya S
Department of Pharmacology, Kyoto University Faculty of Medicine, Japan.
Journal of biological chemistry (UNITED STATES) Sep 5 1988, 263 (25) p12420-6, ISSN 0021-9258 Journal Code: 2985121R
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
The substrate for ADP-ribosyltransferase from Clostridium botulinum was purified from the cytosol of bovine adrenal gland. Purification procedures consisted of ammonium sulfate fractionation, chromatographies on columns of DEAE-Sepharose and phenyl-Sepharose, gel filtration on a TSK-gel G3000SW column, and Mono Q fast protein liquid chromatography. On DEAE-Sepharose chromatography, the substrate activity was eluted in two separate peaks, and electrophoretic analyses revealed that the substrates in the two peaks are of similar molecular weight but different isoelectric points. The major peak of the substrate was further purified. It was purified about 1,800-fold with a recovery of 2.2% by the above procedures. On sodium dodecyl sulfate-polyacrylamide gel electrophoresis, the final preparation showed a single protein band at Mr 22,000. The purified protein served as a substrate for botulinum ADP-ribosyltransferase and was maximally ADP-ribosylated to the extent of about 0.7 mol of ADP-ribose/mol of protein. A guanosine 5'-(3-O-thio)triphosphate (GTP gamma S) binding activity was co-purified with the ADP-ribosylation substrate, and the purified protein maximally bound about 0.5 mol of GTP gamma S/mol. GTP gamma S binding was effectively competed by GTP and GDP but not by GMP, ATP, and ADP. Thus, the ADP-ribosylation substrate is a GTP-binding protein. This protein, designated Gb (b for botulinum), is widely distributed in various tissues. It was rich in brain, pituitary , and adrenal glands, and poor in heart, smooth, and skeletal muscles. Record Date Created: 19880928 Record Date Completed: 19880928

18/7/24 (Item 24 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
09839243 21650335 PMID: 11790300
Rac and Rho mediate opposing hormonal regulation of the ether-a-go-go-related potassium channel.
Storey Nina M; O'Bryan John P; Armstrong David L
Laboratory of Signal Transduction, National Institute of Environmental Health Sciences, Research Triangle Park, NC 27709, USA.
Current biology - CB (England) Jan 8 2002, 12 (1) p27-33, ISSN 0960-9822 Journal Code: 9107782
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
BACKGROUND: Previous studies of ion channel regulation by G proteins have focused on the larger, heterotrimeric GTPases, which are activated by heptahelical membrane receptors. In contrast, studies of the Rho family of smaller, monomeric, Ras-related GTPases, which are activated by cytoplasmic guanine nucleotide exchange factors, have focused on their role in cytoskeletal regulation. RESULTS: Here we demonstrate novel functions for the Rho family GTPases Rac and Rho in the opposing hormonal regulation of voltage-activated, ether-a-go-go-related potassium channels (ERG) in a rat pituitary cell line, GH(4)C(1). The hypothalamic neuropeptide, thyrotropin-releasing hormone (TRH) inhibits ERG channel activity through a PKC-independent process that is blocked by RhoA(19N) and the Clostridium botulinum C3 toxin, which inhibit Rho signaling. The constitutively active, GTPase-deficient mutant of RhoA(63L) rapidly inhibits the channels when the protein is dialysed directly into the cell through the patch pipette, and inhibition persists when the protein is overexpressed. In contrast, GTPase-deficient Rac1(61L) stimulates ERG channel activity. The thyroid hormone triiodothyronine (T3), which antagonizes TRH action in the pituitary , also stimulates ERG channel activity through a rapid process that is blocked by Rac1(17N) and wortmannin but not by RhoA(19N). CONCLUSIONS: Rho stimulation by G(13)-coupled receptors and Rac stimulation by nuclear hormones through PI3-kinase may be general mechanisms for regulating ion channel activity in many cell types. Disruption of these novel signaling cascades is predicted to contribute to several specific human neurological diseases, including epilepsy and deafness. Record Date Created: 20020115 Record Date Completed: 20020327

18/7/26 (Item 26 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
10586395 96401263 PMID: 8807639
Rho proteins are localized with different membrane compartments involved in vesicular trafficking in anterior pituitary cells.
Cussac D; Leblanc P; L'Heritier A; Bertoglio J; Lang P; Kordon C; Enjalbert A; Saltarelli D
I.C.N.E. UMR 9941 CNRS Institut Jean Roche Faculte de Medecine Nord, Marseille, France.
Molecular and cellular endocrinology (IRELAND) May 31 1996, 119 (2) p195-206, ISSN 0303-7207 Journal Code: 7500844
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
In order to explore the role of certain GTP binding proteins in the rat anterior pituitary , we have analyzed the subcellular distribution of the proteins rho and rab. They were found in both membrane and cytosolic fractions. Rab1 and rab2 were localized in both Golgi and endoplasmic reticulum (ER) membranes, while rab4 and rab6 were found in fractions enriched with Golgi and plasma membranes, implicating these proteins in the control of vesicular intracellular trafficking as described in other systems. Rab3 was localized like a fraction of synaptophysin, suggesting a role for rab3 in the targeting of "synaptic-like' microvesicles. We have identified three substrates of C. botulinum exoenzyme C3. A 26-kDa substrate with an isoelectric point (pI) of 5.2, probably rhoB, was localized in the lightest fractions such as rab3 and synaptophysin proteins. Two other 23-24 kDa substrates with pI of 5.5-5.8, probably rhoA and/or rhoC, were found in both fractions enriched with ER and secretory granules. Rho proteins have been implicated in the control of actin polymerization. Their localization in anterior pituitary suggests that rhoB could control the association of synaptic-like microvesicles and plasma membrane, and that rhoA/rhoC could play a role in secretory granule exocytosis; these two pathways being involved in cytoskeleton protein reorganisation in response to extracellular signals. Record Date Created: 19961122 Record Date Completed: 19961122

21/6/1 (Item 1 from file: 155) 03455881 81147314 PMID: 6259462
Acute neuromuscular disorders. Jan 1981

21/6/2 (Item 2 from file: 155) 10037919 21974827 PMID: 11978559
Current therapeutic strategies for hyperhidrosis: a review. May-Jun 2002

21/6/3 (Item 3 from file: 5) 13929174 BIOSIS NO.: 200200557995
Method for treating hypercalcemia. 2002

21/6/4 (Item 4 from file: 5) 14167796 BIOSIS NO.: 200300161825
Method for treating thyroid disorders. 2003

21/6/5 (Item 5 from file: 5) 06291616 BIOSIS NO.: 000086125799
OYSTERS FROM THE MESSINIAN OF ORANIE WESTERN ALGERIA AND PALEOBIOLOGY OF THE WHOLE BIVALVE FAUNA 1988

21/6/6 (Item 6 from file: 155) 10514786 96325713 PMID: 8752407
[Pathophysiologies of dystonia and myoclonus--consideration from the standpoint of treatment] Dec 1995

21/6/7 (Item 7 from file: 5) 05581887 BIOSIS NO.: 000083055027
A SURVEY OF PARASITES OF THE AMERICAN LOBSTER HOMARUS-AMERICANUS CRUSTACEA DECAPODA FROM THE CANADIAN MARITIMES 1986

21/6/8 (Item 8 from file: 155) 11901760 99344722 PMID: 10416233
[Sotos syndrome associated with focal dystonia] Sindrome de Sotos asociado a distonia focal. May 16-31 1999

21/6/9 (Item 9 from file: 155) 15071743 22732198 PMID: 12847738
Type A botulinum toxin: a new treatment for axillary and palmar hyperhidrosis. Sep 2002

23/6/1 (Item 1 from file: 5) 08979593 BIOSIS NO.: 199396131094
Acrylamide disrupts elemental composition and water content of rat tibial nerve: III. Recovery. 1993

23/6/2 (Item 2 from file: 155) 07679354 93134602 PMID: 1336629
Actions of three structurally distinct sea anemone toxins on crustacean and insect sodium channels. Nov 1992

23/6/3 (Item 3 from file: 5) 08756001 BIOSIS NO.: 199395045352
Actions of three structurally distinct sea anemone toxins on crustacean and insect sodium channels. 1992

23/6/4 (Item 4 from file: 155) 10861687 97213149 PMID: 9059914
Anisocoria--a pupillary sign of hippocampal lesions: an experimental study in the cat by using neurotoxins . 1997

23/6/5 (Item 5 from file: 155 10949150 97301645 PMID: 9158046
Arteether: risks of two-week administration in Macaca mulatta. Apr 1997

23/6/6 (Item 6 from file: 5) 10940976 BIOSIS NO.: 199799562121
Arteether: Risks of two-week administration in Macaca mulatta. 1997

23/6/7 (Item 7 from file: 155) 09194522 20503008 PMID: 11048335
Association between malignant tumors of the thyroid gland and exposure to environmental protective and risk factors. Jul-Sep 2000

23/6/8 (Item 8 from file: 155) 04057694 83186924 PMID: 6404956
Brainstem projections to the normal and noradrenergically hyperinnervated trigeminal motor nucleus. Feb 20 1983

23/6/9 (Item 9 from file: 5) 03949972 BIOSIS NO.: 000076035538
BRAIN STEM PROJECTIONS TO THE NORMAL AND NORADRENERGICALLY HYPER INNERVATED TRIGEMINAL MOTOR NUCLEUS 1983

23/6/10 (Item 10 from file: 155) 09091959 20389497 PMID: 10931107
A case of adrenocortical carcinoma associated with recurrence after laparoscopic surgery. Aug 2000

23/6/11 (Item 11 from file: 5) 12658113 BIOSIS NO.: 200000411615
A case of adrenocortical carcinoma associated with recurrence after laparoscopic surgery. 2000

23/6/12 (Item 12 from file: 155) 06214202 89229936 PMID: 2713685
Degeneration patterns in the chicken central nervous system induced by ingestion of the organo-phosphorus delayed neurotoxin tri-ortho- tolyl phosphate. A silver impregnation study. Apr 10 1989

23/6/13 (Item 13 from file: 5) 06701408 BIOSIS NO.: 000088010826
DEGENERATION PATTERNS IN THE CHICKEN CENTRAL NERVOUS SYSTEM INDUCED BY INGESTION OF THE ORGANOPHOSPHORUS DELAYED NEUROTOXIN TRITOLYL PHOSPHATE A SILVER IMPREGNATION STUDY1989

23/6/14 (Item 14 from file: 155) 09790778 21597338 PMID: 11760536
[Depression, stress and brain function] Depression, stress og hjernefunktion. Nov 19 2001

23/6/15 (Item 15 from file: 5) 04721422 BIOSIS NO.: 000080024548
DEVELOPMENT OF SEROTONERGIC AND ADRENERGIC RECEPTORS IN THE RAT SPINAL CORD EFFECTS OF NEONATAL CHEMICAL LESIONS AND HYPERTHYROIDISM 1985

23/6/16 (Item 16 from file: 155) 04894272 85200946 PMID: 2986790
Development of serotonergic and adrenergic receptors in the rat spinal cord: effects of neonatal chemical lesions and hyperthyroidism . Mar 1985

23/6/17 (Item 17 from file: 155) 04924292 85231061 PMID: 3924645
Effects of triiodothyronine and propylthiouracil on regeneration of catecholaminergic nerve terminals in the paraventricular hypothalamic nucleus of the adult rat. Jul 1985

23/6/18 (Item 18 from file: 5) 04766951 BIOSIS NO.: 000080070078
EFFECTS OF TRIIODOTHYRONINE AND PROPYLTHIOURACIL ON REGENERATION OF CATECHOLAMINERGIC NERVE TERMINALS IN THE PARAVENTRICULAR HYPOTHALAMIC NUCLEUS OF THE ADULT RAT 1985

23/6/19 (Item 19 from file: 5) 08988932 BIOSIS NO.: 199396140433
Effects of systemic administration of 6-hydroxydopamine, 6-hydroxydopa and 1-methyl-4-phenyl-1,2,3,6-tetrahydroxypyridine (MPTP) on tuberoinfundibular dopaminergic neurons in the rat. 1993

23/6/20 (Item 20 from file: 155) 11445833 98328939 PMID: 9664237
Evaluation of olfactory and auditory system effects of the antihyperthyroid drug carbimazole in the Long-Evans rat. 1998

23/6/21 (Item 21 from file: 5) 08979548 BIOSIS NO.: 199396131049
Graves' ophthalmopathy and tobacco smoking.1993

23/6/22 (Item 22 from file: 5) 02646402 BIOSIS NO.: 000067034464
Immuno histochemical evidence of substance p-like immuno reactivity in some 5 hydroxy tryptamine containing neurons in the rat central nervous system 1978

23/6/23 (Item 23 from file: 155) 08459898 95148114 PMID: 7845593
Importance of cholinergic, GABAergic, serotonergic and other neurons in the medial medullary reticular formation for sleep-wake states studied by cytotoxic lesions in the cat. Oct 1994

23/6/24 (Item 24 from file: 5 09554656 BIOSIS NO.: 199598009574
Importance of cholinergic, GABAergic, serotonergic and other neurons in the medial medullary reticular formation for sleep-wake states studied by cytotoxic lesions in the cat. 1994

23/6/25 (Item 25 from file: 5) 12538893 BIOSIS NO.: 200000292395
Inhibition of excitotoxic neuronal cell death by total extracts from oriental medicines used for stroke treatment. 2000

23/6/26 (Item 26 from file: 155) 06804013 91043707 PMID: 2562061
Involvement of postsynaptic alpha 2-adrenoceptors and guanine nucleotide-binding protein in guanabenz-induced cardiovascular suppressant effects in the rat. Oct 23 1989

23/6/27 (Item 27 from file: 5) 06868027 BIOSIS NO.: 000089017618
INVOLVEMENT OF POSTSYNAPTIC ALPHA-2-ADRENOCEPTORS AND GUANINE NUCLEOTIDE-BINDING PROTEIN IN GUANABENZ-INDUCED CARDIOVASCULAR SUPPRESSANT EFFECTS IN THE RAT 1989

23/6/28 (Item 28 from file: 155) 06286798 89302949 PMID: 2568126
Isolation, characterization, and amino acid sequence of a polypeptide neurotoxin occurring in the sea anemone Stichodactyla helianthus. Apr 18 1989

23/6/29 (Item 29 from file: 5) 06706616 BIOSIS NO.: 000088016036
ISOLATION CHARACTERIZATION AND AMINO ACID SEQUENCE OF A POLYPEPTIDE NEUROTOXIN OCCURRING IN THE SEA ANEMONE STICHODACTYLA-HELIANTHUS 1989

23/6/30 (Item 30 from file: 155) 01849039 75023799 PMID: 4138314
Letter: Haloperidol, thyrotoxicosis, and neurotoxicity .Oct 26 1974

23/6/31 (Item 31 from file: 155) 01999278 75175148 PMID: 1134541
Letter: Neurotoxic reaction to haloperidol in a thyrotoxic patient.Jul 24 1975

23/6/32 (Item 32 from file: 155) 09568646 21350936 PMID: 11457794
Multiple rostral medullary nuclei can influence breathing in awake goats. Aug 2001

23/6/33 (Item 33 from file: 5) 13184102 BIOSIS NO.: 200100391251
Multiple rostral medullary nuclei can influence breathing in awake goats. 2001

23/6/34 (Item 34 from file: 5) 13929174 BIOSIS NO.: 200200557995
Method for treating hypercalcemia. 2002

23/6/35 (Item 35 from file: 5) 14167796 BIOSIS NO.: 200300161825
Method for treating thyroid disorders. 2003

23/6/36 (Item 36 from file: 155) 11780936 99219419 PMID: 10204725
Nucleus reticularis gigantocellularis and nucleus raphe magnus in the brain stem exert opposite effects on behavioral hyperalgesia and spinal Fos protein expression after peripheral inflammation. Mar 1999

23/6/37 (Item 37 from file: 5) 11969909 BIOSIS NO.: 199900223222
Nucleus reticularis gigantocellularis and nucleus raphe magnus in the brain stem exert opposite effects on behavioral hyperalgesia and spinal Fos protein expression after peripheral inflammation. 1999

23/6/38 (Item 38 from file: 5) 10511662 BIOSIS NO.: 199699132807
Neoplasia and biotoxins in bivalves: Is there a connection? 1996

23/6/39 (Item 39 from file: 155) 14141072 22339658 PMID: 12452498
Neuropathologic toxicity of artemisinin derivatives in a mouse model. Oct 2002

23/6/40 (Item 40 from file: 155) 08039859 94105593 PMID: 1342484
Neurotoxicity caused by cyclosporin A in renal transplantation in children] Neurotoxicidad por ciclosporina A en trasplante renal en ninos. Mar 1992

23/6/41 (Item 41 from file: 155) 05552682 87231765 PMID: 2954144
Neurotoxicity of mitotane therapy of adrenocortical carcinoma (5 cases) and Cushing 's syndrome (7 cases)] Neurotoxicite du traitement par le mitotane des cortico-surrenalomes (5 cas) et des syndromes de Cushing (7 cas). May 23 1987

23/6/42 (Item 42 from file: 5) 05724176 BIOSIS NO.: 000084072582
NEUROTOXICITY OF MITOTANE TREATMENT OF ADRENOCORTICAL CARCINOMA 5 CASES AND CUSHING 'S SYNDROME 7 CASES 1987

23/6/43 (Item 43 from file: 5) 03844686 BIOSIS NO.: 000075022759
PURIFICATION AND PROPERTIES OF A TOXIN FROM THE SEA-ANEMONE CONDYLACTIS-GIGANTEA 1982

23/6/44 (Item 44 from file: 5) 08039275 BIOSIS NO.: 000093072623
PARALYTIC SHELLFISH TOXINS SEQUESTERED BY BIVALVES AS A DEFENSE AGAINST SIPHON-NIPPING FISH 1991

23/6/45 (Item 45 from file: 155) 10673719 97022806 PMID: 8869166
Participation of presynaptic noradrenergic fibers in the suppression of alpha 2-adrenoceptor activity by substance P at the nucleus reticularis gigantocellularis of the rat. Dec 1995

23/6/46 (Item 46 from file: 155) 02031096 75207059 PMID: 167932
Patterns of motoneuron dysfunction and recovery. Feb 1975

23/6/47 (Item 47 from file: 155) 01660142 74065531 PMID: 4766648
Possible potentiation of haloperidol neurotoxicity in acute hyperthyroidism . Nov 1973

23/6/48 (Item 48 from file: 155) 07305495 92168440 PMID: 1686483
Reduction in postsynaptic alpha 2-adrenoceptor activity by endogenous angiotensin III in the nucleus reticularis gigantocellularis of the rat. Nov 25 1991

23/6/49 (Item 49 from file: 5) 07976167 BIOSIS NO.: 000093043745
Reduction In Postsynaptic Alpha-2-Adrenoceptor Activity By Endogenous Angiotensin Iii In Nucleus Reticularis Gigantocellularis Of The Rat 1991

23/6/50 (Item 50 from file: 5) 03356731 BIOSIS NO.: 000072084835
RISK OF APPLICATION OF CONTRAST MEDIUM IN COMPUTED TOMOGRAPHY 1981

23/6/51 (Item 51 from file: 155) 03581498 81273960 PMID: 7022551
[Risk of application of contrast medium in computed tomography (author's transl)] Das Kontrastmittel-Risiko bei der Computer-Tomographie. Jun 1981

23/6/52 (Item 52 from file: 155) 08192581 94258503 PMID: 7515321
Substance P suppresses the activity of alpha 2-adrenoceptors of the nucleus reticularis gigantocellularis involved in cardiovascular regulation in the rat. Feb 28 1994

23/6/53 (Item 53 from file: 5) 09186177 BIOSIS NO.: 199497194547
Substance P suppresses the activity of alpha-2-adrenoceptors of the nucleus reticularis gigantocellularis involved in cardiovascular regulation in the rat. 1994

23/6/54 (Item 54 from file: 5) 13972837 BIOSIS NO.: 200200601658
Stereotactic radiosurgery for patients with ACTH-producing pituitary adenomas after prior adrenalectomy. 2002

23/6/55 (Item 55 from file: 155) 14733314 22527032 PMID: 12640074
Thyrotoxic autoimmune encephalopathy: a repeat positron emission tomography study. A 2003

23/6/56 (Item 56 from file: 155) 02965674 79141708 PMID: 425762
[Toxoplasmosis hypothalamic syndromes in children] Toksoplazmoznye gipotalamicheskie sindromy u detei. 1979

23/6/57 (Item 57 from file: 155) 10029788 21965119 PMID: 11968459
Why do Purkinje cells die so easily after global brain ischemia? Aldolase C, EAAT4, and the cerebellar contribution to posthypoxic myoclonus. 2002

23/7/2 (Item 2 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
07679354 93134602 PMID: 1336629
Actions of three structurally distinct sea anemone toxins on crustacean and insect sodium channels.
Salgado V L; Kem W R
Rohm and Haas Company, Spring House, PA 19477.
Toxicon - official journal of the International Society on Toxinology (ENGLAND) Nov 1992, 30 (11) p1365-81, ISSN 0041-0101
Journal Code: 1307333 Contract/Grant No.: GM 32848; GM; NIGMS Document type: Journal Article
Languages: ENGLISH Main Citation Owner: NLM Record type: Completed

The membrane actions of three recently isolated polypeptide neurotoxins from the sea anemones Stichodactyla helianthus (toxin ShI), Condylactis gigantea (toxin CgII) and Calliactis parasitica (toxin CpI) were investigated on action potentials and voltage-clamp membrane currents of the giant axon of the crayfish Procambarus clarkii. The first two toxins were also tested on the cockroach (Periplaneta americana) giant axon. All three toxins were particularly lethal to crustaceans, moderately toxic to an insect (cockroach), and essentially non-toxic to a mammal (mouse). ShI and CgII were 50- to 100-fold more potent on crayfish than on cockroach axons; this difference in activity was correlated with the relative reversibility of their effects on these arthropod axons. The crustacean selectivity of these toxins is therefore due largely to their greater affinity for crustacean sodium channels. All three toxins prolonged crayfish giant axon action potentials by selectively slowing Na channel inactivation without greatly affecting activation. Before toxin treatment, inactivation was nearly exponential, with a time constant less than 1 msec. After

treatment, the inactivation time course could be described as the sum of two exponentially decaying components, plus a large steady-state component. The major component possessed the slower (10-20 msec) time constant. The steady-state component increased with depolarization, causing the sodium channel steady-state inactivation curve to reach a minimum between -60 and -20 mV and then increase at more positive potentials. All three toxins shifted the peak sodium current-voltage relation to the left. This voltage shift was greater at 20 degrees C than at 10 degrees C. Maintained membrane depolarization during toxin wash-in delayed the appearance of modified Na channels. Also, prolonged depolarization of toxin-treated axons converted modified sodium channels back to normal ones. The toxins did not affect potassium and leakage currents. Our results indicate that the three crustacean-active sea anemone toxins share a common electrophysiological action on arthropod sodium channels, at least at the macroscopic level. Record Date Created: 19930212 Record Date Completed: 19930212

23/7/4 (Item 4 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
10861687 97213149 PMID: 9059914
Anisocoria--a pupillary sign of hippocampal lesions: an experimental study in the cat by using neurotoxins .
Hashida N; Shoumura K; Ichinohe N; Hirama J; Amayasu H
Department of Anatomy, Hirosaki University School of Medicine, Japan.
Journal fur Hirnforschung (GERMANY) 1997, 38 (1) p9-26, ISSN 0021-8359 Journal Code: 0421521
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
Pupillary inequality developed after unilateral lesions of the hippocampal formation (HF) of the cat. Lesions were made by an injection of a small amount of colchicine, ibotenic acid or kainic acid. In all anisoric cats, the pupil on the side of the lesions was invariably smaller than its partner. Evident pupillary inequality developed in the cats with dorsal HF lesions. Although there was a considerable variation in the extent and the location of lesions among these cats, the involvement of the giganto -pyramidal CA 3-2 appeared very responsible for the development of the pupillary sign. However, an evidently anisoric cat hat a lesion affecting only the prosubiculum and the subiculum in the posterior part of the dorsal HF. Despite a large involvement of CA 3-2 with or without coincident injuries to CA1, the prosubiculum and the subiculum, only slight pupillary inequality developed following ventral HF lesions. Evident anisocoria in the cats with large dorsal HF lesions disappeared after bilateral cervical sympathectomy, implying that the asymmetry of sympathetic nervous activity might be critically involved in the development of the pupillary sign. The hippocampo-spinal pathway relayed by the lateral septal nucleus and, then, by the lateral hypothalamic area to terminate in the intermedio-lateral cell column of the spinal cord was considered to be most concerned with anisocoria caused by HF lesions. Record Date Created: 19970522 Record Date Completed: 19970522

23/7/15 (Item 15 from file: 5) DIALOG(R)File 5:Biosis Previews(R) (c) 2003 BIOSIS. All rts. reserv.
04721422 BIOSIS NO.: 000080024548
DEVELOPMENT OF SEROTONERGIC AND ADRENERGIC RECEPTORS IN THE RAT SPINAL CORD EFFECTS OF NEONATAL CHEMICAL LESIONS AND HYPERTHYROIDISM
AUTHOR: LAU C; PYLYPIW A; ROSS L L
AUTHOR ADDRESS: DEP. ANATOMY, MED. COLL. PA., 3200 HENRY AVE., PHILADELPHIA, PA. 19129, USA.
JOURNAL: DEV BRAIN RES 19 (1). 1985. 57-66. 1985 FULL JOURNAL NAME: Developmental Brain Research
CODEN: DBRRD RECORD TYPE: Abstract LANGUAGE: ENGLISH
ABSTRACT: The sympathetic preganglionic neurons in the spinal cord receive dense serotonergic (5-HT) and catecholaminergic (CA) afferent inputs from descending supraspinal pathways. In the rat spinal cord, the levels of these biogenic amines and their receptors are low at birth, but undergo rapid ontogenetic increases in the ensuing 2-3 postnatal wk until the adult levels are reached. In many systems, denervation of presynaptic neurons leads to an up-regulation of the number of postsynaptic receptors. To determine whether the 5-HT and CA receptors in the developing spinal cord are also subject to such transsynaptic regulation, the ontogeny of serotonergic receptors and .alpha.- and .beta.-adrenergic receptors were examined in thoracolumbar spinal cord of rats given neurotoxins which destroy serotonergic (5,7-dihydroxytryptamine (5,7-DHT)) or noradrenergic (6-hydroxydopamine (6-OHDA)) nerve terminals. Intracisternal administration of 5,7-DHT or 6-OHDA at 1 and 6 days of age prevented, respectively, the development of 5-HT and CA levels in the spinal cord. Rats lesioned with 5,7-DHT displayed a marked elevation of 5-HT receptors with a binding of 50% greater than controls at 1 wk and a continuing increase to twice normal by 4 wk. A similar pattern of up-regulation was also detected with the .alpha.-adrenergic receptor, as rats lesioned with 6-OHDA exhibited persistent increases in receptor concentration. In these same animals, ontogeny of the .beta.-adrenergic receptor in the spinal cord remained virtually unaffected by the chemical lesion. In several other parts of the nervous system, the .beta.-adrenergic sensitivity can be modulated by hormonal signals, particularly that of the thyroid hormones. This phenomenon was examined in the spinal cord and in confirmation with previous studies. Neonatal treatment of triiodothyronine (0.1 mg/kg, s.c. daily) was capable of evoking persistent increases in .beta.-adrenergic receptor binding. Development of the postjunctional serotonergic and .alpha.-adrenergic receptors in the rat spinal cord can occur in the absence of the prejunctional nerve terminals and are subject to transsynaptic modulation; .beta.-adrenergic receptors in the spinal cord also can develop after prejunctional lesions but are regulated by hormonal rather than neuronal factors.

23/7/17 (Item 17 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
04924292 85231061 PMID: 3924645
Effects of triiodothyronine and propylthiouracil on regeneration of catecholaminergic nerve terminals in the paraventricular hypothalamic nucleus of the adult rat.
Hoovler D W; Hwang B H; Demers L M
Experimental neurology (UNITED STATES) Jul 1985, 89 (1) p123-33, ISSN 0014-4886 Journal Code: 0370712
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
Effects of triiodothyronine and the antithyroid drug, propylthiouracil, on regeneration of catecholaminergic nerve terminals in the paraventricular hypothalamic nucleus of adult rats were studied. Lesions were produced by 6-hydroxydopamine neurotoxin and then the animals were treated with triiodothyronine or propylthiouracil inducing hyperthyroidism or hypothyroidism, respectively, as determined by radioimmunoassay. Although catecholaminergic varicosities increased with time in the paraventricular hypothalamic nucleus of rats after lesion, fluorescent microscopic quantitation showed no statistical difference in their number between rats treated with triiodothyronine and the vehicle for as long as 56 days. Furthermore, electron microscopic quantitation at 56 days postlesion showed no significant difference between the triiodothyronine-treated and control rats in terms of the density, proportion, size, types of synapses, and synaptic frequency of catecholaminergic nerve terminals. There were growth cones in the paraventricular hypothalamic nucleus, suggesting growth activity after lesion. However, we found that exogenous administration of large doses of triiodothyronine at 25 micrograms/kg had little effect on the enhancement of regeneration of central catecholaminergic terminals after their destruction by 6-hydroxydopamine. Record Date Created: 19850805 Record Date Completed: 19850805

23/7/23 (Item 23 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
08459898 95148114 PMID: 7845593
Importance of cholinergic, GABAergic, serotonergic and other neurons in the medial medullary reticular formation for sleep-wake states studied by cytotoxic lesions in the cat.
Holmes C J; Jones B E
Montreal Neurological Institute, McGill University, Quebec, Canada.
Neuroscience (ENGLAND) Oct 1994, 62 (4) p1179-200, ISSN 0306-4522 Journal Code: 7605074
Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
Previous evidence has suggested that neurons in the medial medullary reticular formation play a critical role in the modulation of forebrain and spinal cord activity that occurs during the sleep-waking cycle and particularly in association with the state of paradoxical sleep. The importance of these neurons, including cholinergic, serotonergic and GABAergic cells [Holmes C. J. et al. (1994) Neuroscience 62, 1155-1178] for sleep-wake states was investigated after their destruction with the neurotoxin quisqualic acid injected into the medullary gigantocellular and magnocellular tegmental fields in cats. To assess the effects of the neuronal loss, polygraphic recording and behavioural observations were performed in baseline and for three weeks after the lesion, and the changes in these measures were correlated with the volume of destruction of medullary regions and the numbers of chemically identified cells within those regions. Following the cytotoxic lesions, which affected approximately 60% of the medullary gigantocellular and magnocellular tegmental fields, there was a significant reduction in the amount of paradoxical sleep (to a mean of 64% of baseline) during the first postlesion week, that recovered variably across cats in the second and third weeks. There was little to no change in the amount or organization of waking and slow wave sleep. The individually variable amounts of postlesion paradoxical sleep were correlated positively with the number of surviving cholinergic cells, negatively with the number of surviving serotonergic cells and positively with the ratio of surviving cholinergic or GABAergic cells to serotonergic cells. The most marked effect of the lesion was a substantial increase in the amplitude of the nuchal electromyogram during slow wave sleep (to 198%) and paradoxical sleep (to 378% of baseline in the first postlesion week). The increase in muscle tone was associated with movements of the head, neck or limbs during paradoxical sleep. Although, in some cats, the abnormal neck muscle tone decreased with time, limb movements continued to occur during paradoxical sleep for the duration of the experiment. The ratio of the total number of remaining cholinergic or GABAergic cells to serotonergic cells correlated negatively with the increased muscle tone and/or movements. It was concluded that the neurons of the medial medullary reticular formation contribute to, but are not necessary for, the generation of paradoxical sleep, and have particular importance for the regulation of muscle tone and inhibition of movement during this state.(ABSTRACT TRUNCATED AT 400 WORDS) Record Date Created: 19950307 Record Date Completed: 19950307

23/7/28 (Item 28 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
06286798 89302949 PMID: 2568126
Isolation, characterization, and amino acid sequence of a polypeptide neurotoxin occurring in the sea anemone Stichodactyla helianthus.
Kem W R; Parten B; Pennington M W; Price D A; Dunn B M
Department of Pharmacology and Therapeutics, University of Florida College of Medicine, Gainesville 32610.
Biochemistry (UNITED STATES) Apr 18 1989, 28 (8) p3483-9, ISSN 0006-2960 Journal Code: 0370623
Contract/Grant No.: GM32848; GM; NIGMS Document type: Journal Article Languages: ENGLISH
Main Citation Owner: NLM Record type: Completed
An aqueous exudate collected from frozen and thawed bodies of a Caribbean sea anemone, Stichodactyla (formerly Stoichactis) helianthus, contained a polypeptide neurotoxin (Sh I) selectively toxic to crustaceans. The polypeptide was purified by G-50 Sephadex, phosphocellulose, and sulfopropyl-Sephadex chromatography and shown to have a molecular size of 5200 daltons and a pI of 8.3. The amino acid sequence determined by automatic Edman degradations of whole RCM Sh I and of its clostripain, staphylococcal protease, and cyanogen bromide digest peptides is A1ACKC5DDEGP10DIRTA15PLTGT20VDLGS25CNAGW30EKCAS35YYTII40ADCCR45KKK . Only 33% of this sequence is

identical with the sequence of Anemonia sulcata toxin II, a sea anemone toxin isolated from the taxonomic family Actiniidae. The six half-cystines are located in equivalent positions to those of the actiniid toxins and account for nearly half of the residues common to all of the toxins. However, 69% of the Sh I sequence is identical with that of toxin II from Heteractis paumotensis, another sea anemone belonging to the family Stichodactylidae. Stichodactylid toxins lack the initial N-terminal residue of actiniid toxins and possess three consecutive acidic residues at positions 6-8, a single tryptophan at position 30, and four consecutive basic residues at positions 45-48 (C-terminus). A rabbit IgG prepared by Sh I immunization bound Sh I with a K0.5 of 4.7 nM but failed to bind homologous actiniid (Anemonia sulcata II, Condylactis gigantea III) or bolocerid (Bolocera tuedae II) polypeptide neurotoxins .(ABSTRACT TRUNCATED AT 250 WORDS) Record Date Created: 19890817 Record Date Completed: 19890817

23/7/39 (Item 39 from file: 155) DIALOG(R)File 155:MEDLINE(R) (c) format only 2003 The Dialog Corp. All rts. reserv.
14141072 22339658 PMID: 12452498
Neuropathologic toxicity of artemisinin derivatives in a mouse model.
Nontprasert Apichart; Pukrittayakamee Sasithon; Dondorp Arjen M; Clemens Ralf; Looareesuwan Somchai; White Nicholas J; et al
Department of Clinical Tropical Medicine and Bangkok Hospital forTropical Diseases, Faculty of Tropical Medicine, Mahidol University,Bangkok, Thailand.
American journal of tropical medicine and hygiene (United States) Oct 2002, 67 (4) p423-9, ISSN 0002-9637 Journal Code: 0370507 Document type: Journal Article Languages: ENGLISH Main Citation Owner: NLM Record type: Completed
Intramuscular administration of high doses of artemether and arteether to experimental mammals produces selective damage to brain stem centers involved predominantly in auditory processing and vestibular reflexes. The relationship between clinical signs of neurotoxicity and neuropathologic toxicity was studied in the mouse. Intramuscular artemether (50-100 mg/kg/day for 28 days) caused dose-dependent neuropathologic damage to the brain stem. There was no pathologic evidence of neuronal death in mice receiving either oral artemether, or oral or intramuscular artesunate, in doses up to 300 mg/kg/day. The neurons in the lower brain stem trapezoid nucleus, the gigantocellular reticular nucleus, and the inferior cerebellar peduncle were the most sensitive to the toxic effects of artemether. All mice with neuropathologic changes also showed behavioral changes, whereas in some mice with gait disturbance, no corresponding histopathologic damage could be detected. Thus clinical assessment was a sensitive measure of neurotoxicity . There may be a reversible component to artemether neurotoxicity . Record Date Created: 20021127 Record Date Completed: 20021212

23/7/43 (Item 43 from file: 5) DIALOG(R)File 5:Biosis Previews(R) c) 2003 BIOSIS. All rts. reserv.
03844686 BIOSIS NO.: 000075022759
PURIFICATION AND PROPERTIES OF A TOXIN FROM THE SEA-ANEMONE CONDYLACTIS- GIGANTEA
AUTHOR: BERNHEIMER A W; AVIGAD L S; LAI C Y
AUTHOR ADDRESS: DEP. OF MICROBIOL., NEW YORK UNIV. SCHOOL OF MED., NEW YORK, N.Y. 10016.
JOURNAL: ARCH BIOCHEM BIOPHYS 214 (2). 1982. 840-845. 1982 FULL JOURNAL NAME: Archives of Biochemistry and Biophysics CODEN: ABBIA RECORD TYPE: Abstract LANGUAGE: ENGLISH
ABSTRACT: A cytolytic toxin from the sea anemone C. gigantea was isolated and characterized as a thermolabile basic protein (pI [isoelectric point] 8.9) having a MW of 18,300. It lacked Met but contained relatively large amounts of Gly, Ser, Trp and Cys. Its hemolytic action was inhibited by sphingomyelin. It was lytic for rabbit blood platelets, lethal in low concentration for crayfish (LD50 = 0.06 .mu.g) and may have been identical with a neurotoxic protein isolated earlier from the same species. It broadly resembled the toxin of Stoichactis helianthus but differed from it in amino acid composition and in minor respects.